

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—22ND YEAR.

SYDNEY, SATURDAY, JUNE 29, 1935.

No. 26.

Table of Contents

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES—	PAGE.	BRITISH MEDICAL ASSOCIATION NEWS—	PAGE.
"Some Early Medical Men of Western Australia", by F. I. BRAY	793	Scientific	818
"The Autonomic Nervous System", by HERBERT J. WILKINSON	801	CORRESPONDENCE—	
REPORTS OF CASES—		The Mortality of Appendicitis	819
"Rupture of the Uterus", by H. A. RIDLER, M.B., Ch.M., F.R.A.C.S.	812	Proprietary Remedies for Injection	820
"Endemic Typhus in New Guinea", by CARL E. M. GUNTHER, M.B., B.S., D.T.M.	813	Medical History of the War	820
REVIEWS—		AUSTRALASIAN MEDICAL PUBLISHING COM- PANY, LIMITED	821
Three Philosophers	814	UNIVERSITY INTELLIGENCE—	
NOTES ON BOOKS, CURRENT JOURNALS AND NEW APPLIANCES—		The University of Sydney	821
An Australian Medical Directory	814	BOOKS RECEIVED	822
LEADING ARTICLES—		DIARY FOR THE MONTH	822
A Twenty-First Birthday	815	MEDICAL APPOINTMENTS VACANT, ETC.	822
CURRENT COMMENT—		MEDICAL APPOINTMENTS: IMPORTANT NOTICE	822
Agranulocytosis	816	EDITORIAL NOTICES	822
The Aetiology of Hypertension	817		

SOME EARLY MEDICAL MEN OF WESTERN AUSTRALIA.

By F. I. BRAY,
State Archives Board, Perth.

DURING the course of the preparation of a paper on "Medical Names in Australian Geographical Nomenclature", eventually printed in THE MEDICAL JOURNAL OF AUSTRALIA, April 28, 1934, Professor J. B. Cleland approached the author for information about early medical men in Western Australia. The information supplied by the author was so extensive and appeared to Professor Cleland to be so valuable that it seemed advisable that it should be kept intact, and not submerged in a more general paper.

CHARLES SMITH BOMPAS was born about 1818. He was admitted a member of the Royal College of

Surgeons, England, in 1840, and a licentiate of the Society of Apothecaries, London, in 1841. Dr. Bompas practised at York in the late fifties and early sixties. He was appointed Resident Medical Officer for the North and stationed at Roebourne on May 25, 1865. About this time he was medical officer to the official party which went north in consequence of the attempt by certain people to settle in Camden Sound (north of 16° latitude). In this regard a company was formed in Melbourne with a capital of £20,000 in £100 shares. The company was known as the Camden Harbour Association. Its declared object was "to settle the very superior well-watered pastoral and agricultural country around Camden Harbour by placing one head of cattle on every 1,000 acres". It was considered by the promoters of the company that £20,000 would secure 4,000,000 acres, and would stock that area with 4,000 breeding cattle.

The company was formed on misleading information as to the character and situation of the country to be settled. An alluring tale was built up as to its potentialities, and as a result shipowners, barristers and other professional and commercial people sought to obtain land. However, the brilliant expectations of the prospective settlers were not realized. The first party of Melbourne settlers arrived in the Colony on December 13, 1864, with sheep, cattle and horses. A more inappropriate season than December, 1864, could probably not have been chosen. It was a time of drought; the heat was intense, sometimes registering before sunset 125° F. in the shade. The party found the land contiguous to Camden Harbour to be parched and destitute of grass and water. Two other vessels of the company with stock and settlers put into Camden Harbour within a few days. Misfortune now came and the sheep began to die off at the rate of 120 a night. The settlers looked for better country, but could not find it. Disaster was then ruthless, and every week's end chronicled further loss of one kind or other. At the end of three months only 1,354 sheep survived; in August all were dead. The pitiless hand of Nature has consummated many such stories, and the pathos of the condition of the immigrants can be imagined. The settlers began to abandon the settlement. Early in 1865 Mr. Robert John Sholl and other government officials, including Dr. Bompas, were sent to the new territory to administer its affairs. Mr. Sholl reported that the company was leaderless; all were masters, none were servants. The sheep had been neglected; they were allowed to wander; were scorched to death by the tropical heat, chilled to death by tropical rains, lamed by sharp burning stones, starved on innutritious grass, killed by native dogs, or lost for ever in the bush. The settlement was abandoned.

Dr. Bompas fell into disagreement with Mr. Sholl and left the north-west. On November 23, 1869, Dr. Bompas was appointed District Medical Officer at Geraldton; House Surgeon, Colonial Hospital Perth, in 1872; Medical Officer at York in May, 1873; Resident Medical Officer at Roebourne in April, 1875; Resident Medical Officer at Vasse in 1879. Dr. Bompas was retired on account of infirmity on April 28, 1886, when he was sixty-eight years of age. His subsequent history is unknown.

ALEXANDER COLLIE was born in Scotland. He voyaged around the world as surgeon of His Majesty's ship *Blossom* before coming to the Swan River settlement in 1829. He arrived here as surgeon on His Majesty's ship *Sulphur*, which ship escorted the *Parmelia* on which the founder of Western Australia (Captain James Stirling, later Sir James Stirling) and the first body of settlers arrived. Both ships arrived at the Swan River in June, 1829. Dr. Collie at once set about exploring, and in November, 1829, discovered the river Collie. The town of Collie now bears his name, and this is important because Collie is now a flourishing coal-mining centre. On July 25, 1830, he submitted a comprehensive report on the causes of sickness, particularly scurvy, on Peel's ill-fated settlement in the neighbourhood of Clarence, and prescribed measures to alleviate it.

Dr. Collie was appointed first Government Resident at Albany in 1831. From April 20 to May 4, 1831, Dr. Collie explored the country between Albany and the French River, and in May he further explored the country near King George Sound. Dr. Collie returned to Perth at the latter end of 1832 to act as Colonial Surgeon, *vice* Dr. Simmons, first Colonial Surgeon, who died with tragic suddenness on October 23, 1831. Dr. Collie died at Albany on November 5, 1835, when about to embark on leave. The cause of death is said to have been asthma, but was probably tuberculosis of the lungs. Dr. Collie was also a naturalist. Some of his letters are said to be in an eastern State's library. About twenty years ago they were published in *The West Australian* through the courtesy of Mr. Walter Gale of Canberra. They are of great interest because they give a most human and sympathetic account of the life of the Colony. More recently, August 19, 1933, an article on Dr. Collie appeared in *The West Australian*.

The early settlers received grants of land according to the value of property brought to the Colony, on the basis of forty acres of land for every £3 of property. Dr. Collie submitted a joint application with Richard Sholl, the purser of the *Sulphur*. In support of this they lodged a list of property containing particulars of six sows, one English pig and six young English pigs, three spades, two pick axes, three rakes and two hoes, and garden seeds to the value of £3. In the application, Dr. Collie disclosed that he was receiving six shillings a day as surgeon on the *Sulphur* and Sholl four shillings a day as purser. They also claimed to have a servant, W. Hamilton (18) who was "qualified to officiate as a hairdresser".

WILLIAM ELGEE was born at Jubbelpore, India, on September 10, 1866. He was a licentiate of the Royal College of Physicians, London, 1890, and a member of the Royal College of Surgeons, England, 1890. He was educated at the Bedford Grammar School, England. He was Clinical Assistant at the Hospital for Sick Children, Great Ormond Street, London. He went through Saint Bartholomew's Hospital and was Assistant House Surgeon at the Liverpool Northern Hospital in 1891. He arrived in Western Australia in 1892, and practised at Guildford and Midland Junction. He was Resident Medical Officer at Perth Public Hospital for three years. He was Mayor of Midland Junction in 1900. He became associated in practice with Dr. J. M. Y. Stewart of Guildford, and died at Midland Junction on March 17, 1906. Dr. Elgee married Amy F. Lee Steere, a daughter of Sir James Lee Steere, for many years Speaker of the Legislative Assembly. Mrs. Elgee is now residing at Park Mansions, Park Street, South Yarra, Victoria.

CHARLES BOLTON ELLIOTT, M.R.C.S. (England), L.R.C.P. (London), was Resident Medical Officer of the Geraldton District from 1874 to 1904, when he died (at Geraldton). He was born in London. He arrived in Western Australia in 1874 in the ship *Naval Brigade*. Immediately on his arrival he was appointed Resident Medical Officer at the Colonial Hospital, Perth. After a residence of six months he was placed in charge of the Victoria District,

pending the arrival of Dr. Rutherford Riley from the north-west. He subsequently returned to Perth for two months, and in December, 1874, as a result of a requisition to the Government from the people of the Victoria District, was permanently appointed to the position. He was also Quarantine Officer, Health Officer, Justice of the Peace, and twice filled the post of Acting Government Resident of the Victoria District. He was Principal Medical Officer of the Western Australia Volunteer Force. When appointed, Dr. Elliott was the only medical man stationed between Perth and Roebourne. In 1889 he represented Western Australia at the Australasian Medical Congress in Melbourne. Dr. C. H. Elliott (no relation) was his predecessor at Geraldton.

Dr. C. B. Elliott's son-in-law (the Reverend P. U. Henn) is at the Guildford Grammar School, Guildford, Western Australia. Mr. Henn married Grace Elliott.

CHARLES HENRY ELLIOTT was admitted a member of the Royal College of Surgeons, England, on March 12, 1858, a licentiate of Midwifery of the Royal College of Surgeons of England on October 12, 1859, and a licentiate of the Society of Apothecaries, London, on October 13, 1859. He was appointed Resident Medical Officer, Colonial Hospital, Perth, in February, 1874; Resident Medical Officer at Champion Bay in 1874; Acting District Medical Officer at Geraldton in 1879; he discharged duties as District Medical Officer at Northampton in 1883; and was District Medical Officer at Champion Bay in 1886. He was succeeded by Dr. C. B. Elliott (no relation). Dr. C. H. Elliott married a Miss Scott, a daughter of Daniel Scott, an early harbour master at Fremantle, and first chairman of the Fremantle Town Council.

ROBERT FAIRBAIRN. Mr. Fairbairn, the magistrate, was not a medical man. His son, Dr. R. C. Fairbairn, resides at 8, Esplanade, Peppermint Grove, Western Australia.

JOHN FERGUSON was born in 1800 and died in 1881. He was admitted a member of the Royal College of Surgeons of Edinburgh in 1822. He was in practice at Auchterlonie in the 1830's, and came to Western Australia in the ship *Trusty*. One of the immigrants under contract to him was William Forrest, father of Lord Forrest. Dr. Ferguson married in Scotland a daughter of Dr. Viveash, who came to Western Australia in the *Britomarte* and settled on the Middle Swan, becoming a Government Resident there. Dr. Ferguson was made a magistrate of the Territory in 1843; he succeeded Dr. Harris as Colonial Surgeon in 1847, and retained the appointment until 1872. He was appointed to the Central Board of Works in 1847; Visiting Medical Officer of Road Parties on the Perth-Fremantle Road in 1852; a member of the Central Vaccine Board in 1861; Immigration Agent and Officer Poor House, Perth, in 1867; and retired in August, 1870, at the age of seventy on a pension of £216 13s. 4d. *per annum*. He claimed to have been the first medical man in Western Australia to use chloroform when he amputated the leg of an aboriginal. Chloroform was first used in Western Australia in 1849. In 1859 Dr. Ferguson bought the Houghton Vineyards, which are still conducted by his son Mr. C. W. S.

Ferguson, now in his eighty-fifth year. An article on "John Ferguson" appeared in *The West Australian* of July 15, 1933. It was written by the Reverend Canon Burton, of The Rectory, Nedlands, Western Australia.

JOHN STEPHEN HAMPTON obtained the diploma of the Royal College of Surgeons, Edinburgh, in September, 1828. He was for some time on active service as a surgeon in the Royal Navy, his home port being Portsmouth, and on several occasions was placed in charge of the transport of convicts. For some time he served in the Colonial Office at Westminster, and subsequently held the appointment of Comptroller-General of Convicts in Tasmania, where he was known as a severe disciplinarian. Later on he returned to the Colonial Office at Westminster, and was then appointed Governor of Western Australia, which appointment he held from February 28, 1862, to November, 1868. Governor Hampton arrived at Fremantle on February 27, 1862, by the ship *Strathallen*. It was anticipated, because of his wide experience in convict administration, that he would institute radical and beneficial changes in the public works policy of Western Australia, and that the best results would be obtained from convict labour. His term of office justified the expectations. He almost immediately infused new spirit into public works. He increased the number of labourers engaged in erecting Government House, in metalling streets, and in building the Swan River wall. In 1863, when Governor Hampton had surmounted the onus of the debt left him by Governor Kennedy, strong parties were appropriated to work in Perth and suburbs, ninety men were stationed on the North Fremantle road, excavations for the site of the North Fremantle bridge were hurried forward, and a bridge and court house at Geraldton were finished. The plans for Government House were found to be very unsuitable, and Governor Hampton caused extensive alterations to be made in 1863, before the structure was finished in 1864. About the same time a marine summer residence was erected for the Governor at Rottnest Island. In 1863 he commenced the work of erecting the Pensioners' Barracks on the declivity of Mount Eliza, and these are now occupied by the Public Works Department. In Governor Hampton's address to the Legislative Council in June, 1864, appears the following passage, which conveys the best indication of the times:

The public works now in progress are the bridges at North Fremantle, Perth Causeway, Greenough Flats, Beverley and Ferguson River; jetties at Bunbury and Busselton; additions to the Poor House, Perth, and the Police Stations at Pinjarra and Newcastle; new police stations at Baylup, Staunton Springs, and the Lakes; and the reconstruction of the swamp drain at the back of Perth. Forty-nine road parties are distributed in different districts. With the exception of the Pinjarra Police Station and the two jetties, all the works are carried on by convict labour, which costs the colony literally nothing for supervision and maintenance, or for the tools used by them, and the material benefit thus derived by Western Australia from transportation is further enhanced by upwards of £90,000 Imperial money being expended annually in the colony.

It was decided early in 1867 to erect a town hall in Perth by convict labour, and on May 24 the corner stone of that prominent building was laid by Governor Hampton with appropriate ceremony. Governor

Hampton compiled special regulations for the north-west country, subject to the approval of the Imperial Government, and offered Mr. Padbury, or any other person actually settled there, the use of 100,000 acres, in 20,000 acre blocks, for the term of twelve years, the first four years to be without rent. The lands were divided into classes and special conditions applied to each class. The administration of Governor Hampton was significant for an important change—the inauguration of a semi-elective form of government. When it became known that the transportation of convicts was to cease, colonists considered that they were justified in asking to be allowed to elect representatives to the Legislative Council. This view was approved by the British Government and authority was received for the appointment of non-official members equal in number to the official members. Subsequently six gentlemen were elected by the people for nomination by Governor Hampton, and on July 7, 1868, an Order-in-Council was passed constituting them members of the Legislative Council for three years. On December 27, 1867, Governor Hampton recommended that at the end of the first term of three years half the Council be elected, the other half to consist of official members, and that the Governor have a casting vote. The Secretary for State, on March 27, 1868, gave his approval to the recommendation.

Governor Hampton established the Post Office Savings Bank in Western Australia, initiated the money order system, saw the Roman Catholic cathedral open and the Congregationalists erect their first place of worship, laid the foundation stone of Wesley Church, and completed the building of Government House. He despatched an expedition (under Surveyor C. C. Hunt) which passed over the site of Coolgardie without glimpsing any gold, but discovered Hampton Plains; and he welcomed the first wool clip from the north-west—a paltry seven bales—which arrived at Fremantle. In his last year of office, with a population of 25,000 and a cultivated area of only 50,000 acres, he showed an excess of income over expenditure of £10,000. And when he laid down his office of Governor of Western Australia, and left its shores in 1868, he left it (incredible as it appears in these days of staggering national burdens) absolutely free of public debt.

Governor Hampton's tenure of office closed in November, 1868, when he left the colony. His administration was appreciated. As early as August, 1867, a memorial was sent to the Secretary of State for the Colonies, signed by hundreds of persons, praying for the continuance of Governor Hampton's term of office. The heads of the Civil and Military Departments, followed by a long procession of carriages, escorted him to Fremantle on November 2, 1868, where a demonstrating farewelling was made.

He retired in order to return to England owing to the serious health of his wife, who required treatment unobtainable in Western Australia. But his wife died on arrival in England, and he then returned to Torquay. When he died he was buried in Paddington, London, and it is said that his death was really the outcome of an accident sustained some years previously when he slipped on the stairway of Government House, Perth, through the pedestal of a brass lamp which

lighted the stairway giving away under the pressure of his hand.

A grandson, Mr. H. G. Hampton, occupies the position of Under Secretary for Law in Western Australia.

JOSEPH HARRIS (not Joseph Strelley—Joseph Strelley Harris was not a medical man) was appointed a Magistrate of the Territory on December 30, 1837. He was appointed Acting Colonial Surgeon on February 10, 1843, and Colonial Surgeon on July 25, 1845, which position he occupied until his death in 1847. George Fletcher Moore, in his book "Ten Years of Western Australia", wrote on March 3, 1834: "Dr., Mrs. and Miss Harris called". Dr. Harris pursued farming and pastoral pursuits on the Upper Swan. Moore again wrote on May 13, 1837, that Harris had told him of his discovery of the junction of the Hotham and the Williams Rivers to form the Murray River. Sir George Grey in the same year met Dr. Harris, and states he was the first man to overland sheep from Albany to the Swan. Dr. Harris succeeded Dr. James Crichton (1836–1842) as Colonial Surgeon.

According to Neilson Hancock, Registrar of the Medical Board of Western Australia, the son of Dr. Harris and the late J. B. Rowe (a Sheriff of Western Australia) played together as children. At this time the galvanic battery was introduced to Western Australia, and Rowe and young Harris used to be interested to see frogs getting electric shocks. Mr. Hancock understands that Dr. Harris lived opposite the present Government House at Perth in a little cottage which was used at one time as the Colonial Hospital. The cottage is now numbered 22, Saint George's Terrace, and was at one time occupied by Dr. Lance A. Hayward.

RUSTAT HENRY HEMSTED, M.D. (Brussels), M.R.C.S. (England), L.R.C.P. (London), was a son of Mr. Henry Hemsted, surgeon of Whitechurch, Hampshire, England, his grandfather and great-grandfather also having followed the same profession, while two of his brothers are doctors and two dentists. Born at Whitechurch on December 25, 1868, Dr. Hemsted received his early scholastic training at Epsom College, subsequently taking his medical diploma from Saint Mary's Hospital, London, in 1893. After practising for some time at Wakefield, in Yorkshire, he joined his father in practice in his native town, and at a later date entered into partnership with Dr. Arthur Coates, of Bristol Infirmary, where he spent eleven years. The more genial climate of Cornwall tempted him to that county for a time, and the same reason prevailed in eventually bringing him to Australia in 1910, reports of the favourable climatic conditions, especially of the western State, having reached him in the old country. Among the appointments held in England by Dr. Hemsted (who is a member of the British Medical Association and also a Life Member of the Clinical Research Association, London) were those of District Medical Officer and Medical Officer to the Infirmary, Whitechurch; Deputy Medical Officer to the Isolation Hospital; Surgeon to the Police and Post-Office; and other appointments in the Public Service. Later he became Clinical Assistant at the London Throat

Hospital, and also studied in Brussels, where he obtained the degree of Doctor of Medicine at the University of that city. At the beginning of 1910 he decided to give up practice for a year and to devote his time to the study of the recent developments in medicine and surgery, for which purpose he attended some of the chief London hospitals, including the West London; the Hospital for Diseases of the Ear, Nose and Throat, Golden Square; and the London Throat Hospital. Upon arrival in Western Australia he proceeded to Geraldton, where he established himself in his profession. He has been a keen motorist for many years, and was one of the first doctors in England to use a motor car in place of horses, this hobby with fly-fishing constituting his principal amusements. He married in 1898 Gertrude, daughter of Mr. W. Munn, who for many years was in charge of the Bank of England note mills, and now lives in retirement. There were two daughters of the marriage, one being now Mrs. W. E. Dempster of Cooralya Station, Carnarvon, Western Australia. In 1920 Dr. Hemsted left Geraldton after nine years' practice there, and returned to England for a holiday and to take up further post-graduate work in medicine and surgery at the West London Hospital, and nose and throat work at the Gray's Inn Road Hospital for those complaints. Returning in 1921 to Western Australia he was appointed Medical Officer to the Carnarvon District and Hospital with the usual appointments. This he retained for a period of five years. During the time he was in Carnarvon he had the misfortune to lose his wife. He then returned to Perth to live in semi-retirement. In 1928 he married, secondly, Daisy, the only daughter of the late William Fletcher, solicitor, of Sheffield, England. Dr. Hemsted is the eldest son of the late Henry Hemsted of Whitechurch, Hampshire, and represents the sixth generation of his family in unbroken succession to practise medicine (perhaps a record). Dr. Hemsted resides at 41, Kanimbla Road, Hollywood, Western Australia.

Dr. Edmond Spencer Hemsted is a brother of Dr. H. M. Hemsted. The former practises in Newbury and in Knitburgh, where he has a residence.

The Hemsted family as medical practitioners have been well represented in the county of Berkshire for several generations, Dr. Stephen Hemsted, of Ilsley, having been specially popular as a doctor and sportsman. His son, Lieutenant Charles Hemsted of the Royal Navy, was Governor of the Naval Knights of Windsor.

JOHN SYDNEY HICKS, L.S.A., 1887, M.B. (London), 1888, M.D. (London), 1890, was the son of the late James Sampson Hicks, of Falmouth, Cornwall, where he was born in 1864. He received his primary education at the Falmouth Grammar School, matriculating at the London University. Upon obtaining his medical diploma, he was appointed House Surgeon and Physician to the London Hospital in 1889, and continued to fill the post for about a year, during which period he took his M.D. degree. In 1890 Dr. Hicks left for Western Australia, and upon arrival in the Colony proceeded to Roebourne to succeed Dr. Kelsall as District Medical Officer,

remaining there until 1898. In that year he took a trip to England, and upon his return in 1900 turned his attention to public life, being elected unopposed for the Roebourne seat in the State Legislative Assembly. He was reelected in 1904 and again returned in September, 1905, subsequently holding the portfolio of Minister for Labour and Commerce in the Rason Government from August 25, 1905, to May 7, 1906, when he resigned with the Government. After resigning his portfolio, Dr. Hicks continued in the Assembly as a private member until 1908, when he declined further nomination. He established a private practice at Guildford in 1906, and continued there for many years. He was a member of the Medical Board of Western Australia and also occupied a seat on the Midwifery Board, the special function of which is to deal with the registration of midwives. He was fond of shooting, and found recreation chiefly in the exercise of his skill in that direction. In 1899 he married Margaret, daughter of the late Captain R. R. Pearce, of Cornwall, England, and had a son and a daughter. The date of Dr. Hicks's death is unknown.

HINES. Nothing definite can be ascertained about a medical man named Hines. L. C. Burges, in his diary, refers to a Dr. Hinds and to a Mr. Hinds. Burges states that he dined with Hinds on July 21, 1841. Dr. Cyril Bryan states that Hinds was settled on the Upper or Middle Swan in 1841. Dr. Cyril Bryan is a reliable authority on the early medical history of Western Australia, so far as it is known. There is a record of a person named C. R. Hinds being resident here in 1839 to 1844. He was appointed a Magistrate of the Territory on April 12, 1844. It is not known, however, whether or not he was a medical man.

FREDERICK MAURICE HOUSE, M.R.C.S. (England), 1888, L.R.C.P. (London), 1888, was born at "Privett", Alverstoke, England, on July 16, 1865. He went through Saint Thomas's, London, in 1890. He came to Western Australia in 1891 and was appointed District Medical Officer, Beverley, on January 1, 1892; District Medical Officer at Katanning on March 1, 1893; District Medical Officer at Derby in November, 1895; and District Medical Officer at Beverley on November 1, 1899. In 1899 Mount House was named in his honour by Frank Hann the explorer. He was naturalist to the government party despatched in April, 1901, under the leadership of F. S. Brockman, with Mr. Charles Crossland as second in command, to explore the extreme northern end of the State, lying between the seventeenth and fourteenth parallels of latitude, and west from the one hundred and twenty-eighth meridian. To use Mr. Brockman's own description of his trip: Leaving the port of Wyndham on May 9, the party proceeded in a southerly direction, following the course of a previously unexplored river (named the Chamberlain) to the seventeenth parallel and proceeded thence westerly, principally over high sandstone tablelands, to the Charnley River, which had been explored and named by Mr. F. Hann in 1899. Mr. Hann's position of this river and the neighbouring features were found to be geographically accurate. The party then traced the Charnley and

Isdell Rivers westerly from Hann's exploration to their respective points of exit in tidal waters. They also traced the course of the Sale River and tributaries (discovered by Mr. T. C. Sholl in 1865, but placed too far south by him), and the course of the Glenelg River (discovered by Sir George Grey in 1837, and also previously shown in error of latitude). They discovered and traced the course of the Calder River, and the headwaters of the Prince Regent River; located the positions of the tidal waters extending inland from Collier and Doubtful Bays; and generally investigated the country lying to the south-west and south-east of the main watershed, which is situate about the sixteenth parallel of latitude, and to the westward of the one hundred and twenty-sixth meridian. From the northern fall of this watershed the Roe River was traced from its source to its exit into the tidal waters of Prince Frederick Harbour. The Moran River was discovered, and its course between the same points traced. The headwaters of the King Edward River were discovered at the watershed, and this river was again picked up in about latitude $15^{\circ} 15'$, and its course traced northerly to its exit into Napier Broome Bay. Portions of the shores of Admiralty Gulf and Vansittart and Napier Broome Bays were closely examined with a view to selecting a suitable port for the district. The Drysdale was traversed from its mouth (on the fourteenth parallel of latitude) to the main watershed previously referred to on the sixteenth parallel. At the same time a sufficient number of points on the Carson River were located to enable that stream to be mapped with approximate accuracy. The Durack River was traced from the seventeenth parallel of latitude to its entrance into the tidal waters of Cambridge Gulf. The whole of the country drained by the rivers already enumerated was investigated as closely as practicable in an exploration of this description, all high points met with being ascended, and short excursions being made at right angles to the main line of travel wherever practicable. The exploration was completed on November 20 by the arrival of the leader and his party at the Pentecost River, at a point previously fixed by him on May 18. The practical results of the expedition consisted of the discovery of a large area (six million acres) of basaltic pastoral country covered with blue grass, Mitchell and kangaroo grasses, and many varieties of top feed, lying principally in the neighbourhood of the Charnley, Calder, Sale, Roe, Moran and Carson Rivers, with some extensive areas in addition situated on the Drysdale, and in similar patches in the neighbourhood of the Durack and its tributaries. In addition the existence of suitable ports and routes of access to enable this country to be utilized for stock raising were ascertained. Many objects of scientific interest amongst the flora and fauna of the district were discovered and brought back by the party. A few aboriginal weapons and implements, and a large number of photographs of curious cave paintings were obtained. A considerable amount of information was also obtained with regard to the numbers, habits and distribution of the aborigines of the country.

On this expedition Dr. House discovered the black grass wren (*Amytornis housei*). The bird is

recorded only in exceedingly rough country strewn with masses of sandstone, and this gives some idea of the country traversed by the expedition. Dr. House resumed duty as District Medical Officer and Resident Magistrate at Katanning on September 24, 1902. He visited England from April to September, 1909, and enlisted in the Australian Imperial Force on August 1, 1915, serving two years in Egypt under Colonel Matthews. On his return from the War Dr. House resigned from the Public Service on December 31, 1918, and devoted himself to his stud sheep farm at "Privett", Gnowangerup. He has established there a merino sheep stud which has become notable throughout Western Australia. He has won on several occasions the highest honours at the Royal Agricultural Show at Claremont, Western Australia, and there is no doubt that his professional knowledge has been of great assistance to him in connexion with his success in merino sheep breeding. Unfortunately Dr. House is now very ill.

ADAM JAMESON, Bachelor of Medicine, Master in Surgery, University of Edinburgh, 1883, was born in Fifeshire, Scotland, about 1860. He was the second son of a Presbyterian minister. While a student he suffered from cadaveric poisoning which permanently impaired his health and made it necessary for him to live in a warm and equable climate. About 1885 he came to Western Australia and worked either as an assistant or partner with Dr. Harvey, a noted practitioner. About 1888 he married Miss Ethel Hensman, only daughter of Mr. Justice Hensman, of Perth. In or about 1890 they went to Europe with a view to living in London, but Dr. Jameson found the climate too trying for his health and eventually he entered into partnership with a Dr. Thomson in Rome, Italy. Mrs. Jameson died in Rome in 1897, and in that year, or the next, Dr. Jameson returned to Perth, with his three young daughters, and set up a joint establishment with the Hensmans. His practice was chiefly that of a consultant, although he visited former patients and old friends. Dr. Jameson was elected to the Legislative Council as a member for the Metropolitan Province on August 29, 1900. He was Minister without portfolio in the first Leake Government from June 28, 1901, to November 21, 1901, and was appointed Minister for Lands in the second Leake Government, November 24, 1901. The Leake Government resigned on July 1, 1902, but Dr. Jameson continued to hold the same office in the James Government from July 1, 1902, to January 23, 1903. In Western Australia Dr. Jameson met Sir Arthur Lawley (later Lord Wenloch), Governor of Western Australia, 1901-1902, and the latter subsequently, when Lieutenant-Governor of the Transvaal, invited him to occupy the position of Commissioner of Crown Lands, Transvaal, which position he took up in 1903. He retired on an Imperial pension when the Union of South Africa was created, and decided to travel back to England via Portuguese South Africa. Unfortunately the train was wrecked by a washed-out culvert, and Dr. Jameson was killed while asleep in his berth.

Charles Jameson is a brother of Dr. Jameson. He resides in or near Brisbane. Charles Jameson

was a judge for many years in Brisbane, and is now over eighty years of age.

Mrs. Griffiths-Foulkes, a daughter of Dr. Jameson, resides at 1, Bolton Gardens, Kennington, England.

HENRY TRUMAN KELSALL, a general practitioner, Perth, was registered under the Imperial Act. He was a Master in Surgery, University of London (1885), member of the Royal College of Surgeons (1886), licentiate of the Royal College of Physicians (London) (1887). He took his degree of Doctor of Medicine of the University of London in 1888 with honours. He was President of the Medical Board of Western Australia, and died whilst holding that office. Dr. Kelsall was born at Nani Tal, India, on June 4, 1865, and was a son of the late Surgeon-Major Kelsall. Dr. Kelsall was educated at the United Service College, Westward-Ho, England, and while there was a friend and contemporary of Mr. Rudyard Kipling, with whom he corresponded after he came to Western Australia. After leaving school Dr. Kelsall entered the London University and gained his Doctor of Medicine degree and obtained his hospital experience in the London Hospital, being clinical assistant to Sir Frederick Treves and Sir Andrew Clarke. He served later in the Moorfields Ophthalmic Hospital, where he was house surgeon to John Whittaker Hulke. As a medical student he was a great friend of Sir Wilfred Grenfell, of Labrador, with whom he maintained regular correspondence until his death. He worked for some years with Sir Wilfred as a medical officer to deep-sea fishermen. Subsequently, Dr. Kelsall joined the Royal Navy as a surgeon, but in 1890 he resigned and migrated to Western Australia, taking up a practice at Roebourne. Dr. Kelsall took over the appointment of Acting Resident Medical Officer at Roebourne from his friend of student days, Dr. T. Frizell. Dr. Frizell resigned the appointment, but before doing so he wrote to Kelsall advising him of his intention, and thereby enabled Kelsall to apply for the position. Subsequently Dr. Kelsall removed to Perth and took up a general practice, and later became an ophthalmic specialist. From 1892 to 1917, when he retired, he was honorary ophthalmic surgeon to the Perth Hospital, of which he was also a member of the first board of management. With the late Sir Winthrop Hackett, he was a member of the Acclimatization Committee of Western Australia, which for many years did the work now performed by the Government at the Zoological Gardens at South Perth. For years he was a trustee of the Museum, Public Library and Art Gallery. After retiring, Dr. Kelsall lived in the Moora District. He became interested in the Berkshire Valley, east of Moora, where one of his sons took charge in 1925 on the father's return to Perth. Dr. Kelsall was a keen sportsman, having played "A" grade standard cricket, tennis and hockey. He was at one time captain of the West Perth cricket team, and also a member of the once famous Wanderers cricket club, being a familiar figure when opening the innings with the late Ernest Parker, a great cricketer who was killed in the Great War. Dr. Kelsall played pennant tennis for different clubs around Perth, notably the Wanda and Claremont clubs. He was for years President of the Perth Rifle Club, having

been a good shot with both rifle and gun, and was also a keen fisherman. In 1892 the late Dr. Kelsall married Miss Blanche Edith Leake, daughter of the late Judge Leake and sister to the late George Leake, Premier of Western Australia from May 27, 1901, to November 21, 1901, and from December 23, 1901, to July 1, 1902. Dr. Kelsall died on May 19, 1932, and is buried in the Karrakatta cemetery.

Mrs. H. T. Kelsall resides at Bay View Terrace, Buckland Hill, Western Australia.

HENRY LANDOR was a brother of E. W. Landor, an early Western Australian lawyer, magistrate and author of "The Bushman", which he published in 1847. At page 196 of that publication there is a chapter entitled "Remarks on the Physical Organization of the Natives" with a footnote stating that "the observations in this chapter were contributed by Henry Landor Esq., Colonial Surgeon on the Gold Coast, who resided five years among the natives of Western Australia and is intimately acquainted with all their habits". At page 9 the author mentions that he was accompanied to Western Australia by two brothers, and at page 266 he states "the doctor arrived from York". This latter evidence that Henry Landor was a doctor and resident at York links up with official records which reveal that Henry Landor was appointed a Magistrate of the Territory on November 26, 1841, and a Justice at York on April 26, 1844.

Henry Landor became interested in the belief that an inland sea existed and he and a friend, H. M. Lefroy, were ambitious to discover the mysterious water. On January 9, 1843, they left York with a native named Cowit to look for it in the country to the south and south-east; but, though they struggled over scores of miles under exceedingly great difficulties, they found no sea. Cowit was supposed to act as interpreter and to shoot kangaroos. For ten miles the little party held to the Albany Road; then they turned south-east into the Corbiding country. From Corbiding they proceeded to a place called by the natives Nymbatilling, and on to the Hotham River. Through rain and rough country they searched. First they examined Carbal, a fertile valley beyond Narjaling, with its grassy floors trending for miles. Lake Byriering was reached on January 14, and round about were lakes so numerous that they might well think they were on the borders of an inland sea. From a hill the explorers observed a treeless plain of sand and scrub and a lake studded with islands in varied forms; they had never seen land and water so tastefully mingled. Eastwards they passed a series of salt lakes—Norrington, Quiliding, Byriering, Quabing, Barkiering, Quiliwhirring, Goondering, Damoeling and others. A river came upon was named Landor; a pool, the Cowit, and a second river, Lefroy. Hot scrubby repellent plains upon which was no water, and a wretched class of country generally were surveyed, and a point on the Williams River was reached. From the Williams River they returned to York.

There is no record of Henry Landor being here in 1886 or 1887, and the medical lists for those years do not mention him.

CHARLES WILLIAM LAVER was born at Castlemaine, Victoria. He was admitted a licentiate of the Royal College of Physicians and a licentiate of the Royal College of Surgeons of Edinburgh in 1894, a licentiate of Midwifery, Edinburgh and Glasgow, in 1894, and a licentiate of the Fellowship of Physicians and Surgeons of Glasgow in 1894. Inspired by the thrilling accounts of the explorations in the Kimberleys of the late Alexander Forrest (brother to Lord Forrest) in 1879-1880, Dr. Laver decided to come to Western Australia, and arrived here in the early eighties. He was then in his teens and too young to start his medical course at the Melbourne University. On reaching Perth, Dr. Laver secured, through Mr. Forrest's help, some of the best pastoral country on the Fitzroy and Margaret Rivers, some 250 miles inland towards Hall's Creek in the Kimberleys. It was on the first visit to Perth in the early eighties that Dr. Laver saw and handled the very first piece of gold found in Western Australia, a nugget the size of a hen's egg, which had been found on a marsh near Roebourne in 1881. It was shown to Dr. Laver at the Weld Club, Perth, by the Honourable W. E. Marmion and Sir Thomas Cockburn Campbell, Members of the Legislative Council, and by Lord Forrest (then Surveyor-General) and his brother Alexander Forrest. Three years later, when prospecting his pastoral country in the Kimberleys, the first gold rush in Western Australia broke out at Hall's Creek, near his holding, and he secured one of the first alluvial nuggets on the first goldfields in Western Australia in 1885-1886. This nugget he still possesses. It is the shape of a map of England and weights about five pennyweights. It is strange how history repeats itself. Ten years later, when pioneering the British Flag District, Laverton, he got what was then the farthest inland alluvial nugget of gold in Western Australia of five ounces, which was also the shape of the map of England. This latter nugget was found on the British Flag Settlement, which was owned by Wilkie Brothers, the noted railway constructors, and Dr. Laver.

Dr. Laver's first return trip from the Kimberleys in 1883 was made in a small pearling schooner of thirty tons, which had been chartered by Mr. Julius Brockman, one of Australia's finest bushmen, and a member of the well-known Western Australian family of that name who was one of the first pioneers to bring sheep to the Kimberleys in the schooner. Coming out of King's Sound (Derby), where the eddies are powerful, agitated and noisy, for the tide has a rise and fall of thirty-six feet, the little boat whirled around three times in one huge eddy before it shot out to safety, much to the relief of the party. The little boat took four weeks to reach Cossack, which should have been accomplished in one week or less. For three weeks the food of the party consisted mainly of turtle, which the party secured on the ninety-mile beach. In due course Cossack was reached and the boat beached, and she was still there when Dr. Laver visited Marble Bar thirty years afterwards.

After a jaunt out to the Lachlan and the Darling, along the famous seventy-mile track, and on the heads of the far-off Paroo, Barcoo and Warrego

Rivers, Dr. Laver resumed his studies and finalized at Edinburgh. He studied also in Paris and, later, was in Professor and Madame Curie's laboratory when Madame Curie had just isolated the first piece of radium which he saw. On returning to Melbourne in 1894, Dr. Laver put up his plate next to the Melbourne Club, in Collins Street. Then the thrill of the Coolgardie rush claimed him and he left for Western Australia. Upon reaching Southern Cross he rode horseback from there to Coolgardie, and on to the "Six Mile" and Slug Hill rushes, which had just broken out. At this time the first shaft of the Great Boulder was started and about a week afterwards Dr. Laver and others inspected it to its then depth of ninety feet. On reaching the bottom it was found that the men had just struck ore glittering with gold in a drive going south. It looked like spongy retorted gold. The first bucket of golden ore was brought up and it yielded 264 ounces of gold. Up to 1929 some £17,000,000 worth of gold had been taken out of the Great Boulder.

Proceeding to Lake Carey, Mount Margaret, Dr. Laver camped on the site of Sir John Forrest's farthest east camp of 1869, alongside of a waterhole at the foot of Mount Margaret. Dr. Laver was so impressed with what he saw there that he resolved to remain, and at once secured a personal interest in the British Flag claim. Subsequently the town of Laverton was named in honour of Dr. Laver, in recognition of his many services to the locality.

The British Flag claim was the pioneer goldmine of the district. It was owned by Wilkie Brothers and Dr. Laver. It produced near the surface 1,000 ounces from five tons of stone, and a total of £25,000 worth of specimens; yet the deepest shaft today is only 100 feet. Mr. John Wilkie, of New Zealand, and Dr. Laver are now further testing the property. Subsequently, in 1906, other mines were opened up at Laverton, the Craigie More, which produced half a million sterling, the Ida H., which produced a million and a quarter, and the Lancefield, which produced over a million.

At Laverton the aborigines showed Dr. Laver the striking picture of the emu and the kangaroo in the Southern Cross and Milky Way constellation.

In the Laverton district Dr. Laver walked (on losing his camel) fifty miles in one day between sunrise and sunset. He also rode a bicycle 120 miles between sunrise and sunset, and on one occasion was in the saddle for 100 miles in the day, using a fresh horse every thirty miles.

Typhoid fever on the goldfields was very severe, and Dr. Laver saw many touching scenes and endings. He found prospectors walking about in the bush (ambulatory typhoid) with high temperatures, delirious and demented, getting no treatment or attention. Some were found doing such things as "reading brown paper", or crawling about on hands and knees, looking for a grave to get into. One suffered from the delusion that he could feel green sprouts growing out all over him; another fancied he was galloping on horseback, trying to free himself of a woman's head (cut off and under his arm and the hair streaming for miles). Typhoid victims were

taken to the Coolgardie cemetery almost as passengers might be taken in cabs to a railway station.

Dr. Laver was a friend of Carr-Boyd, the well-known bushman and explorer, one of nature's gentlemen, whose extraordinary and exaggerated stories never harmed anyone. When in London, Carr-Boyd and Dr. Laver had the honour of being invited to an entertainment at the Savage Club. At such entertainments everything in the nature of music, songs and anecdotes had to be original. Carr-Boyd "played" on the gum leaf for them. He related how, when he was in the wilderness of Western Australia exploring, and ran short of food, he was forced to eat steaks from his living pack camels to save his life; the parts, he said, would heal up again. He told them how, when perishing for the want of water, he discarded his clothes, and saved his life by letting the tropical sun blister his skin, and then moistening his tongue and lips with the blister "water", and how crows had disclosed to him the whereabouts of water in the scrub by calling out "Carr Carr". When bootless, he "shod" himself with tacks from his pack saddles. He told of how bushmen became able, by force of habit, to do with little water, like camels. One bushman, meeting another on a dry stage, and inviting him to have a drink out of the waterbag, might receive a reply such as: "Thanks, old man; but you have not much left. I had a drink only yesterday."

Recently Mr. Sam Hazlett, the veteran Western Australian bushman, with his son, two natives and twelve camels, returned ostensibly from a prospecting trip, but really from an unsuccessful search in unexplored desert for an iron box, wagon tyres and bullocks' horns, the supposed relics of the ill-fated Leichhardt expedition. The search had its origin in the theory of Dr. Laver, who has not accepted the varied conclusions as to the fate of the expedition. In Dr. Laver's opinion, it is probable that the bones of the expedition will be found in some unexplored part of the desert country on the borders of Western Australia and South Australia. Long ago Dr. Laver communicated his ideas to Mr. Hazlett, who has spent the best part of thirty years prospecting the country east of Laverton. Mr. Hazlett lost no opportunities of making inquiries among the natives, to all of whom he is known by name and whose language he speaks. Well aware as anyone of the utter unreliability of native reports, he satisfied himself by the coincidence of details from various sources and by careful cross-questioning that some relics of a party existed in an unexplored part of the desert, but as previously stated he did not succeed in locating them.

Dr. Laver is a brother of the late Frank Laver, one of the greatest of Australian cricketers. Another brother is Professor W. Laver, of Melbourne, the Ormond Professor of Music. Dr. Laver himself accompanied the Darling team of 1899 to England.

Dr. Laver resides at "Bonnie Doon", Kalgoorlie.

HENRY N. WOLLASTON was the third son of John Ramsden Wollaston, first Archdeacon of Western Australia, a notable clergyman. Henry was born on December 14, 1822. His father reached Western Australia in 1841 with his wife and family, but Henry

remained in England studying. He passed his first examination at Apothecaries Hall on March 21, 1840, and followed the others to Western Australia, arriving at Fremantle in the *Janet* on April 30, 1843. He appears to have acted as medical adviser on board during the voyage, and his skill was made use of by the settlers in Western Australia. According to the *Government Gazette* he was appointed Medical Attendant at Albany on February 5, 1850, and Kimberley's "History of Western Australia" states (page 158) that he was appointed medical officer to the convicts in the Albany district in July, 1851. On February 4, 1851, he was appointed a Magistrate of the Territory. Henry Wollaston married a Miss Sewell. He resided at Bicton, near Bunbury, and remained there after his father (Archdeacon Wollaston) removed to Albany, but later on went to Melbourne, where he became a clergyman at Trinity Church, East Melbourne. Sir Harry Wollaston was a son of Henry Wollaston. Sir Harry was born at Mokine, Western Australia, on January 17, 1845, and became Comptroller-General and Permanent Head of the Department of Trade and Customs in 1901. Sir Harry was a Doctor of Laws. Particulars of his most interesting career are given in "John's Notable Australians".

THE AUTONOMIC NERVOUS SYSTEM.¹

By HERBERT J. WILKINSON,
Professor of Anatomy, Adelaide University.

THE principal object of my talk this evening is to give a concise account of the anatomy of the autonomic nervous system so that you, as medical practitioners, may be able to follow more easily the literature for which a present-day knowledge of this system is an essential background.

The main facts concerning the autonomic nervous system have been known since the publication of the classical works of Gaskell (from 1885) and of Langley (from 1900), but it is only quite recently that this system has attracted the attention of clinicians. This has stimulated research into the finer details of the system, not only with regard to its peripheral, but also to its central, connexions within the brain and spinal cord; and a considerable volume of literature has therefore appeared in the last few years.

As it would have been impossible in the short time at my disposal this evening to review the whole of this literature, I approached one or two of your members for suggestions as to what aspect of the subject they would like me to present, and it was decided that it would perhaps be best to develop the subject as a general introduction, so that those interested could approach the new literature with more confidence.

So that the main facts may be the more readily assimilated, I have embodied them in a series of

¹ Read at a meeting of the South Australian Branch of the British Medical Association on March 28, 1935.

simple diagrams which have been prepared for teaching purposes here in the Anatomy School. Some of these have been adapted from well known figures, while others are original, and all suffer from the usual defects inherent in diagrams of complex mechanisms, but have been chosen with the hope that the subject will be made as clear and concise as possible.

The nervous system as a whole is nowadays roughly divided into two parts—the “somatic” or “cerebro-spinal” system, and the “autonomic” or “vegetative” nervous system. The former is that concerned with the reaction of the body to its external environment, and is more or less under voluntary control, while the latter is an automatic mechanism responsible for the control of the internal economy of the body.

Structurally these two systems are more or less superimposed on one another, each consisting of a central and a peripheral part. The central parts of the two systems together form the central nervous system (central organ or cerebro-spinal axis), consisting of the brain and spinal cord, and the peripheral parts include all the nerves, nerve plexuses and nerve ganglia, macroscopic as well as microscopic, which are found throughout the body outside of the central nervous system.

The peripheral parts consist of afferent and efferent conductors which carry impulses to and from the central organ. The afferent conductors arise in terminal organs or receptors which are capable of responding or reacting to the various appropriate stimuli that fall upon them, while the efferent conductors carry impulses from the central organ outwards to effectors. The ultimate somatic effectors are striated muscle fibres in voluntary muscles, while those of the autonomic consist of the glandular epithelial cells and the unstriated plain muscle cells throughout the body in the blood vessels, various viscera *et cetera*.

As far as is known, all afferent conductors have their cells of origin in the posterior root ganglia or their cranial equivalents, namely, ganglia of the roots of the cranial sensory nerves, and they therefore consist of one neurone.

All somatic efferent conductors arise from the anterior cornual cells of the spinal cord or from the cells of origin of the motor cranial nerves. The central organ, therefore, exerts its influence on the somatic effectors throughout the body via paths which consist of only one neurone, the cell body of which is situated in the central organ itself (see Figure V, left side).

In the case of the autonomic nervous system the efferent path consists of two neurones (see Figure V, right side, and Figure II). The cell bodies of the first or proximal neurones are situated in the central organ near those for the somatic efferents, while those of the second or distal neurones are situated in ganglia belonging to the peripheral nervous system. The proximal neurones are the connector cells and give rise to medullated pre-ganglionic fibres. These leave the central organ in company with the somatic efferent fibres via the spinal and

the cranial motor nerves, and end in some ganglion or other before reaching the autonomic effectors to which they discharge their impulses. These ganglia contain the cell bodies of the distal neurones in the autonomic path and give rise to post-ganglionic fibres (usually fine non-medullated fibres), which pass to the effectors. The cells of these ganglia are thus called excitor cells. The autonomic efferent path therefore consists of: (i) connector cells in the central organ, and their medullated pre-ganglionic fibres, and (ii) excitor cells in peripheral ganglia and their fine, usually non-medullated, post-ganglionic fibres.

Although for descriptive purposes we have considered the nervous system as being composed of two more or less superimposed systems, namely, the somatic and autonomic, we must always keep in mind that when impulses are discharged from the central organ through the somatic efferents, impulses are at the same time discharged through the autonomic efferent conductors. There can be no action in the somatic system without coincident action in the autonomic system. The two systems are intimately associated, both structurally and functionally, so that there can be no adjustment of the body as a whole to its external environment through the somatic system without there being associated phenomena due to the automatic activity of the autonomic nervous system.

INNERVATION OF EFFECTOR MECHANISMS.

As stated above, the effector mechanisms are of two kinds, namely, somatic and autonomic, the somatic being the anatomical muscles, for example, *musculus biceps brachii et cetera*, and the autonomic the various viscera, for example, heart, lungs, stomach and alimentary canal generally, bladder, salivary glands *et cetera*.

All effector mechanisms, both somatic and autonomic, can be considered as consisting of two parts, namely, (i) the essential tissue, contractile or glandular, and (ii) the blood circulatory mechanism, and both these parts receive their separate nerve supply. All effector mechanisms can therefore be regarded as having a double nerve supply, one set innervating the essential tissue, and the other the blood vessels.

In the case of the somatic effectors (see Figure I), we have the somatic motor nerves which supply the striated muscle fibres, and the vasomotor nerves which supply the vessels.

In the case of the autonomic effectors, we have one set of autonomic fibres which supplies the contractile elements (that is, plain muscle cells), or the glandular epithelial cells, and another set which supplies the vessels. The former set of autonomic fibres mostly emerges from the central nervous system, from the brain stem and the sacral region of the spinal cord, while the vasomotor nerves mostly arise in the thoracic and upper lumbar regions. It is usual, therefore, to describe the autonomic system as consisting of a cranio-sacral outflow or parasympathetic system, and a thoraco-lumbar outflow

or sympathetic system (see Figure II). Fibres from each of these systems supply each autonomic effector, and the action of the one is antagonistic in effect to that of the other; hence the names, sympathetic and parasympathetic.

These facts may be summarized in Table I.

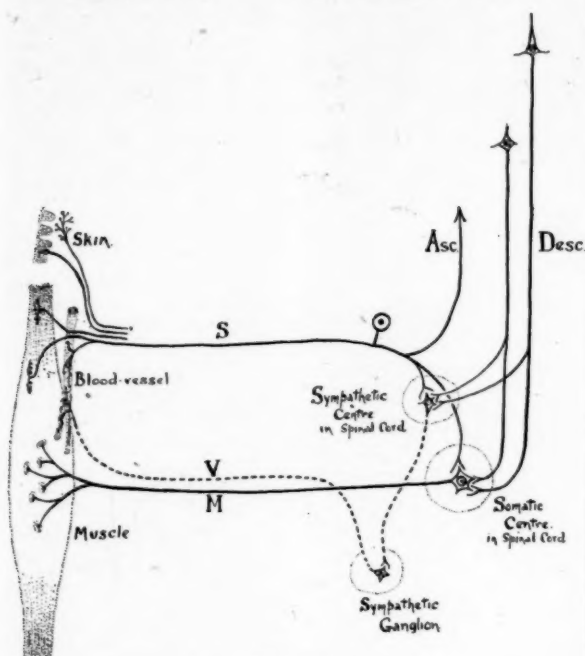


FIGURE I.

Innervation of a somatic muscle.

Figure II is a diagram showing the fundamental division of the autonomic system. On the left side is shown the cranio-sacral outflow (parasympathetic), and on the right the thoraco-lumbar (sympathetic). It may be mentioned that this division of the peripheral autonomic system is not absolute, as will be shown later, but it is convenient for descriptive purposes.

We shall now consider each division in more detail.

THE CRANIO-SACRAL AUTONOMIC OUTFLOW OR PARASYMPATHETIC SYSTEM.

Figure III shows the parasympathetic system in greater detail and should be referred to in the following description.

The connector cells of this system are situated in: (i) the nucleus of Edinger-Westphal (a group of small cells in the nucleus of the third cranial nerve),

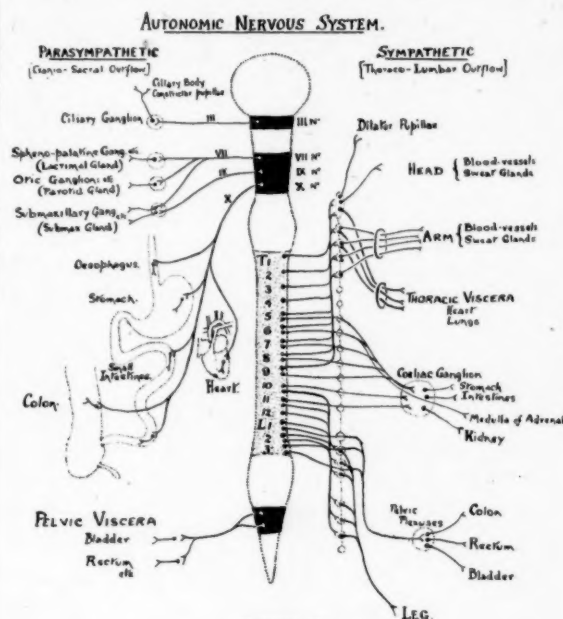


FIGURE II.

General plan of autonomic system (after Langdon Brown).

(ii) the *nucleus salivatorius superior* (similar group in cranial nerve VII), (iii) the *nucleus salivatorius inferior* (similar group in cranial nerve IX), (iv) the dorsal nucleus of the vagus (X), and (v) groups of small cells in the second and third (or the third and fourth) sacral segments of the spinal cord.

From these connector cells preganglionic fibres pass out of the central nervous system along with the somatic conductors, and for the most part do

TABLE I.

Effector Mechanisms.		Composition.	Innervation.
Somatic	Voluntary somatic muscles	(a) Striated muscle fibres (b) Plain muscle cells of vessels	Somatic efferents (anterior cornual cells) Sympathetic efferents (always)
Autonomic	(1) Viscera	(a) Plain muscle cells of organ (b) Plain muscle cells of vessels	Parasympathetic efferents (usually ¹) Sympathetic efferents (usually ²)
	(2) Glands	(a) Glandular epithelial cells (b) Plain muscle cells of vessels	Parasympathetic efferents (usually ²) Sympathetic efferents

¹ Exceptions: Heart and certain sphincters of the hollow viscera (see below), where innervation is reversed.

² Sweat glands in the skin are innervated by sympathetic efferents.

not terminate until they reach the organ they supply, where they end in relation to excitor cells. These excitor cells are grouped together mostly in microscopic ganglia which are situated actually in the organ and cannot be displayed by dissection. They can be easily demonstrated, however, in microscopic sections of the heart, lungs, alimentary canal, salivary glands, bladder *et cetera*. Those in the alimentary canal are known as the ganglia of Auerbach's myenteric and Miessner's submucous plexuses. Some of the cranial preganglionic parasympathetic fibres end in macroscopic ganglia and form an exception to the above rule. These ganglia are the ciliary, sphenopalatine, otic and submaxillary ganglia, and their relations to the cranial nerves are shown in the figure. However, all the excitor cells connected with the cranial outflow are not situated in these ganglia, for sections of the corneo-sclerotic junction of the eye-ball, of the numerous salivary glands, named and unnamed, and of the lachrymal gland, reveal the presence of numerous microscopic ganglia which contain further excitor cells. The latter are probably far more numerous than those found in the four named cranial ganglia.

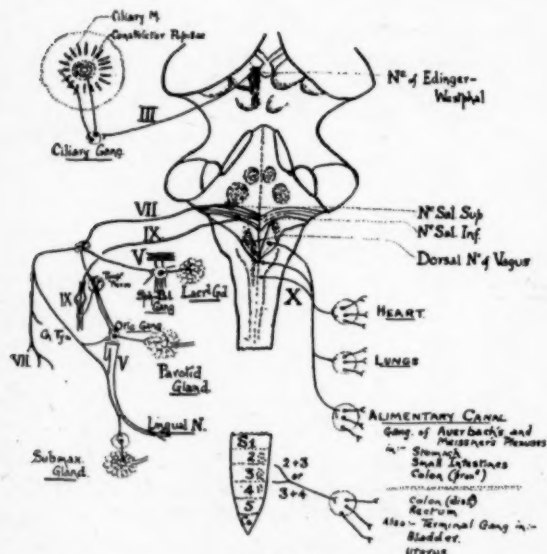


FIGURE III.

Cranio-sacral outflow or parasympathetic system.

The excitor cells in the various terminal ganglia of the parasympathetic then give rise to short post-ganglionic fibres which give off numerous branches; the terminal endings of these branches lie in relation to the plain muscle or epithelial cells which they innervate. The terminations are pericellular and not intracellular, and probably act on the effector cells through the liberation of a chemical substance (see below).

The cranial part of the parasympathetic outflow, through the vagus, innervates the viscera probably as far down as the middle of the transverse colon,

whereas the sphere of influence of the sacral parasympathetic is restricted to the pelvic viscera and external genitalia.

THE THORACO-LUMBAR AUTONOMIC OUTFLOW OR SYMPATHETIC SYSTEM.

Figure IV shows the sympathetic system in greater detail and should be referred to in the following description.

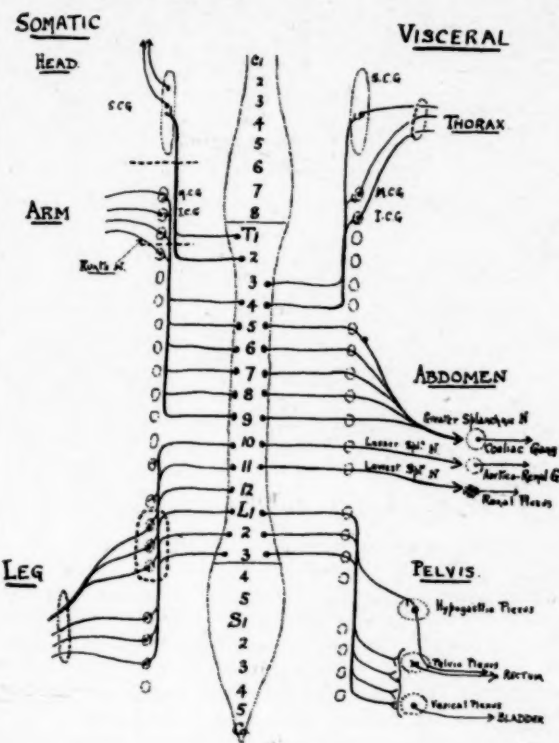


FIGURE IV.

Lumbo-sacral outflow or sympathetic system.

All the connector cells for the sympathetic system are situated in the lateral cornu of the thoracic and upper lumbar segments of the spinal cord, namely, the first thoracic to the second or third lumbar segments.

The pre-ganglionic fibres pass out of the cord via the anterior roots and the white *rami communicantes* to the sympathetic chain of ganglia. Those fibres belonging to the sympathetic paths which are destined for the somatic parts of the body as distinct from the visceral, end here around excitor cells. These in turn give rise to post-ganglionic fibres which return via grey *rami communicantes* to the appropriate spinal nerve with which they are distributed in the periphery (see Figure V, right side). The exact path for sympathetic impulses to the somatic effectors of the head, arm and leg are shown on the left-hand side of the diagram (Figure IV). The pathways for somatic effectors in the trunk are not shown for fear of making the diagram too complicated and difficult to follow. Their arrangement

is more or less segmental, the excitor cells being situated in that ganglion of the sympathetic chain belonging to the segment supplied.

The ganglionated sympathetic chain contains three cervical, ten to twelve thoracic, four lumbar and four sacral ganglia, and all contain excitor cells which are concerned principally with the sympathetic innervation of somatic structures. It also contains, however, those which supply the thoracic viscera (see Figure IV).

The excitor cells for the abdominal and pelvic viscera are situated in more distal ganglia which are to be found in the ganglionated plexuses on the surface of the aorta and its branches. The pre-ganglionic fibres reach these via the various splanchnic nerves after passing through the sympathetic chain (see Figure IV).

The Splanchnic Nerves.

There are two or three named splanchnic nerves and several that are unnamed. The largest or greater splanchnic nerve is formed by branches of the fifth to the ninth or tenth thoracic sympathetic ganglia. These branches are collected together into one large nerve trunk which pierces the crus of the diaphragm and ends in the celiac ganglion. On the course of the greater splanchnic nerve there is a ganglion called the splanchnic ganglion, from which, and from the nerve itself, branches pass to the oesophagus and thoracic aorta (see Cunningham's Text Book, sixth edition, Figure 642, page 790).

The lesser splanchnic nerve arises in the sympathetic chain opposite the ninth and tenth ganglia and passes over the bodies of the lower thoracic vertebrae, pierces the crus of the diaphragm near to or in company with the greater splanchnic nerve, and ends in the aortico-renal ganglion.

There is sometimes a third or lowest splanchnic nerve, which arises from the last thoracic ganglion or as a branch of the lesser splanchnic. This pierces the diaphragm and ends in the ganglionated plexus on the renal artery (renal plexus).

From the lumbar and sacral sympathetic ganglia other unnamed visceral or splanchnic nerves arise and pass to the various abdominal plexuses. These can best be described by reference to Figure 645, page 797, in Cunningham's Text Book, sixth edition. We here see the aortic and the inferior mesenteric plexuses situated on the anterior aspect of the abdominal aorta. The inferior mesenteric plexus is very small and sometimes indistinguishable in man, but is quite large in some lower animals. The aortic plexus gives rise to several nerves which descend into the pelvis along the back and front of its bifurcation and the proximal parts of the common iliac arteries. These nerves are sometimes collected together into one trunk (pre-sacral nerve), but they end in the hypogastric plexus, which lies on the pelvic surface of the body of the first sacral vertebra. This plexus is continued on each side of the rectum (right and left pelvic and hæmorrhoidal plexuses), and forwards to the bladder (vesical plexus) and the genitalia (cavernous, uterine plexuses *et cetera*).

These various plexuses are seen to receive branches from the lumbar and sacral ganglia of the sympathetic.

Two branches to the pelvic plexus are seen to arise from the second and third sacral nerves and not from ganglia of the sympathetic chain. These contain pre-ganglionic fibres of the sacral parasympathetic system and are on their way to the pelvic organs, where they end in terminal ganglia around parasympathetic excitor cells.

There are several other subordinate sympathetic plexuses found on the branches of the aorta, such as the superior mesenteric plexus, the phrenic on the inferior phrenic artery, the suprarenal, spermatic, plexus of the ovarian artery *et cetera*.

The Rami Communicantes.

Mention was made just now of the white and grey *rami communicantes* of the spinal nerves (see Figure V). The white rami are the branches of the anterior spinal nerve roots of the thoracic and upper two or three spinal nerves, and connect the segments of the spinal cord from which these nerves arise with the sympathetic chain. They contain the medullated pre-ganglionic fibres of the sympathetic connector cells, which are situated only in these segments of the cord. They are white because they contain medullated nerve fibres, and at the most they can only be fifteen in number.

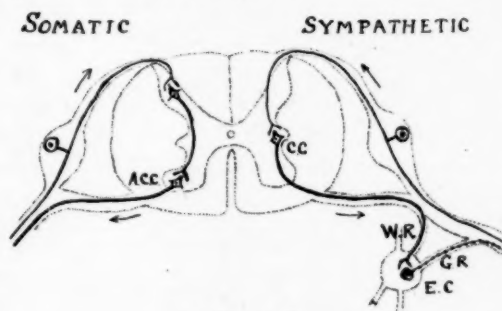


FIGURE V.
(See text.)

The grey rami convey the post-ganglionic fibres of the excitor cells in the ganglia of the sympathetic chain back to the spinal nerves, with which they are distributed to the periphery. They are grey because they are composed mostly of fine non-medullated fibres. The grey rami are much more numerous than the white, as two or more pass to every spinal nerve. In connexion with the sympathetic innervation of the arm, there are as many as twelve to seventeen grey rami which join the branches of the brachial plexus. These run in different directions and in different planes in different individuals.

Sympathetic Denervation of Somatic Structures.

The exact situation of the connector cells and excitor cells in the sympathetic pathways to the head, neck, arm and leg are shown in Figure IV and need no further description. The site of surgical section of the pathways necessary to denervate com-

pletely these parts as far as the sympathetic is concerned are also indicated. To denervate the head, for example, it is only necessary to cut the sympathetic trunk below the superior cervical ganglion. For the arm the trunk is cut between the first and second thoracic ganglia. Arising from the second thoracic ganglion there is a nerve (Kuntz's nerve) which connects with the brachial nerves, and this should be cut also if complete denervation is to be effected.

For the leg, the lumbar ganglia are excised as indicated.

It will be noticed by reference to Figure IV that the section necessary for the arm also denervates the head, neck and thoracic viscera, and that necessary for the leg cuts off the sympathetic supply to the pelvic viscera.

Sympathetic Denervation of the Viscera.

As the sympathetic nerves reach the various viscera via the plexuses on their arteries of supply, a viscus can be denervated by stripping the plexus from the wall of its artery. Sometimes a grosser denervation, for example, of the pelvic viscera, is effected by cutting the hypogastric (pre-sacral) nerves. This will not effect a complete pelvic sympathetic denervation, because of the sympathetic connexions through the sacral sympathetic ganglia.

The exact situation of excitor cells for the various pelvic viscera has not yet been mapped out with complete accuracy; neither is it possible to supply information which will enable a surgeon to cut with certainty all the sympathetic pathways to any one particular pelvic viscus while leaving the innervation of all other viscera intact.

The Central Mechanism of the Autonomic Nervous System.

It has been known for a long time that the autonomic connector cells situated in the central nervous system are under the influence of higher centres. In connexion with the thoraco-lumbar (sympathetic) outflow, for example, there has been found in the hind-brain (*medulla oblongata*) such centres as vaso-constrictor, vaso-dilator, cardio-accelerator *et cetera*, as well as centres for carbohydrate metabolism, for the suprarenal mechanism, for dilatation of the pupil *et cetera*.

More recently the brilliant researches of Le Gros Clark and others have revealed the presence of still higher centres in the fore-brain, which preside over the activity of the autonomic system and through which this activity is correlated and coordinated with that of the somatic system.

Le Gros Clark has undertaken an extensive research into the comparative anatomy of the diencephalon and its relation with the cortex and brain-stem, and the following is a summary of some of his results. It might be well, however, to pause for a moment so as to clear up what may be difficulties to some of you in the nomenclature.

You are probably aware of the fact that the brain and spinal cord are derived embryologically from a simple tube, the anterior end of which undergoes

expansions to form the brain, while the posterior end, which is associated with the cervical, thoracic, lumbar and sacral segments of the body, develops into the spinal cord. At an early stage the anterior end exhibits three dilatations or vesicles which eventually give rise to the fore-brain, mid-brain and hind-brain respectively (see Figure VI). The hind-brain consists of the cerebellum, pons and *medulla oblongata*, the mid-brain is the part around the aqueduct of Sylvius, and the fore-brain is all the rest of the brain in front of this.

In development the fore-brain vesicle develops two lateral evaginations or secondary vesicles, which give rise to the cerebral hemispheres, or end-brain, and the cavity of which becomes the lateral ventricles. The unevaginated part of the fore-brain becomes hidden by the cerebral hemispheres and occupies a space below and between them; hence its name, 'tween-brain or diencephalon. The cavity of the diencephalon is the third ventricle. This region, then, was the field of the research undertaken by Le Gros Clark.

Some of the facts as revealed by his researches can be summarized in the following way:

DIENCEPHALON.

PARS DORSALIS. Somatic correlation mechanism.

Epithalamus: Pineal or epiphysis.

Habenula (related to the olfactory parts of the end-brain).

Thalamus.

Upper Level:

Dorso-medial nucleus \longleftrightarrow Pre-frontal areas of cortex.

Lateral nuclei \longleftrightarrow Association areas of cortex.

Lower Level:

Ventral nucleus proper \longleftrightarrow Tactile sensory projection area. (Receives "sensory" fibres conveying impulses of spinal and trigeminal origin *et cetera*.)

Lateral geniculate body \longleftrightarrow Visual sensory projection area. (Receives optic fibres.)

Medial geniculate body \longleftrightarrow Auditory sensory projection area. (Receives auditory fibres.)

Anterior nucleus \longleftrightarrow Cingulate area. (Receives fibres conveying impulses of olfactory origin.)

PARS VENTRALIS. Visceral correlation mechanism.

Hypothalamus. Including:

Pars nervosa hypophysis or posterior lobe of pituitary. (This plus anterior lobe of pituitary forms a neuro-glandular mechanism.)

Interpeduncular nucleus }
Subthalamic nucleus of Luys } \rightarrow { Red nucleus
Diffuse cells of *zona incerta* } { *Substantia nigra*
Reticular nuclei

Subthalamus. Including:

(This forms a series of relay stations for fibres which descend from the *corpus striatum* and are essential elements in the extrapyramidal system.)

You notice that Le Gros Clark divides the diencephalon into two parts: a dorsal part, which is concerned with the reception and correlation of somatic afferent impulses, and a ventral part, similarly concerned with visceral afferent impulses. These two parts are intimately connected to provide further correlation between the somatic and visceral mechanisms at this level.

The dorsal part contains the thalamus. The ventral part of the thalamus receives impulses from all the somatic receptor organs of touch, pain, temperature, sight, hearing, smell and taste, and is reciprocally connected with the sensory reception areas in the cerebral cortex, while the rest of it is reciprocally connected with the silent association

and prefrontal areas of the cortex. In the evolutionary series of vertebrates these two parts of the thalamus develop in association with the areas of

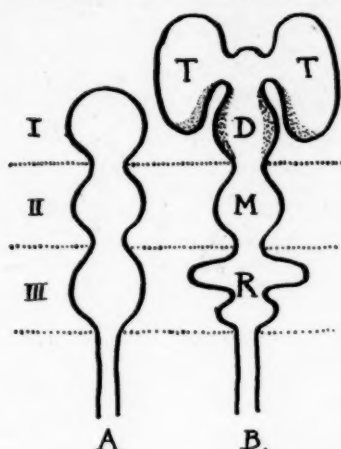


FIGURE VI.

A. Early stage in the development of the neural tube—the three primary vesicles of the brain. B. Later stage, showing the differentiation of the first primary vesicle into T, the telencephalon or end-brain (cerebral vesicles) and D, the diencephalon or 'tween-brain.

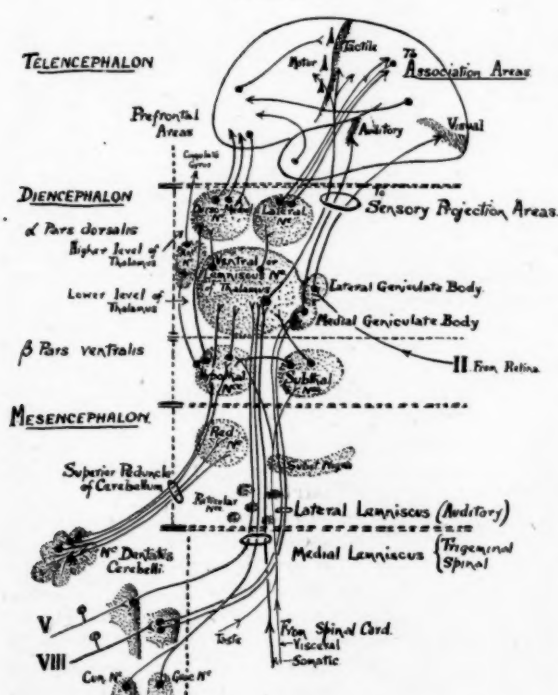


FIGURE VII.

Some connexions of diencephalon.

the cortex with which they are connected, so that the latter part is phylogenetically more recent than the ventral part; it is in fact a primate acquisition.

The ventral part of the diencephalon contains many centres grouped collectively under the designations of hypothalamus and subthalamus. The hypothalamic group is in the lower half of the wall of the third ventricle and is especially associated with the pituitary, forming a neuro-glandular mechanism.

The subthalamus is lateral to the hypothalamus and represents a group of centres which form part of a series of relay stations for fibres which descend from the *corpus striatum* in what is known as the extra-pyramidal system of descending pathways.

Figures VII and VIII are attempts to show some of the connexions of the diencephalon. In these, for the sake of simplicity, the pathways for smell and taste impulses are omitted. As these sense organs can be regarded as both somatic and visceral, we find that impulses which arise in them reach both

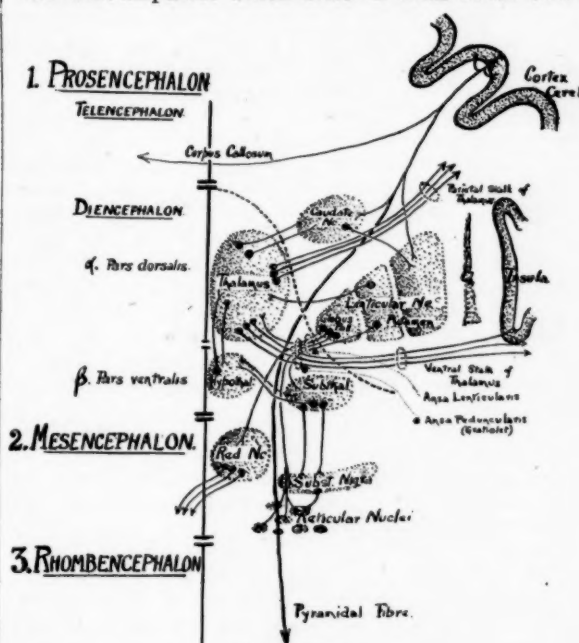


FIGURE VIII.

Some connexions of diencephalon and basal ganglia.

the thalamus and the hypothalamus. Figure VIII shows some of the descending paths, including some of the connexions of the basal ganglia of the end-brain.

This recent morphological research, which has established the significance of the ventral part of the diencephalon as an important central mechanism associated with the activity of the visceral or autonomic nervous system, has been to some extent confirmed by the recent experimental physiological research of Beattie and others, and also by recent clinical investigations.

Time will not permit me to give a review of the physiological experiments, but you will perhaps be interested to hear of Penfield's case as an example of clinical confirmation of the morphological and physiological findings. (The following account is taken from Gask and Ross.)

This was the case of a woman who suffered repeated attacks of fits. A fit started by her asking for a piece of ice to suck. Her face and arms became flushed and red, the respiration was slowed, tears streamed from both eyes, there was sweating and salivation, her pupils dilated, and her eye-balls sometimes protruded. The pulse at first was strong and rapid; then the flush faded and the pulse became weak and slow. The woman would then hiccup a few times, would shiver slightly, and the breathing would become irregular and sometimes goose-flesh would appear on the arms. Finally the attacks were sometimes followed by constipation and inability to empty the bladder. At autopsy a tumour of the chorioid plexus was found hanging in the anterior-superior part of the third ventricle and protruding into the foramen of Munro and producing internal hydrocephalus. There was also oedema in the medial anterior and superior aspect of the thalamus on each side. Histologically the oedematous areas show fading out of the cell structure and some local hyperæmia.

It is significant that the tumour was situated close to the diencephalic centres controlling the visceral activities of the body and that the clinical manifestations described by Penfield correspond to effects produced by stimulation of the autonomic nervous system. The intermittent nature of the symptoms was no doubt due to the free motility of the tumour.

Again, Fulton and others have recently described what they call the syndrome of the premotor cortex. Their results are based on clinical observations and on experiments done on monkeys, and they state that in lesions restricted to the premotor area of the cortex they notice, among other things, changes in skin temperature and sweating on the side of the lesion, and they give this as evidence of representation of the autonomic system in the cortex.

We thus see that above the brain stem centres in the *medulla oblongata* there are centres in the diencephalon and cortex for the correlation and integration of visceral impulses and for the control of autonomic activity. There is thus a hierarchy of centres in the central nervous system for the autonomic system, just as there is for the somatic system.

Figure IX is an attempt to represent this more graphically in diagrammatic form. Starting at the lower levels of the hierarchy, we see that stimulation of one post-ganglionic fibre activates merely the group of gland or plain muscle cells with which it terminates, while stimulation of a pre-ganglionic fibre exerts a wider influence, and a still wider effect is produced by stimulation of the spinal centres. The *medulla oblongata* then controls the whole mechanism and causes the secretion of adrenaline, thus enhancing the total effect of stimulation of the sympathetic. The diencephalon coordinates the afferent impulses and is intimately associated with the hypophysis, stimulation of which leads to secretion of hormones, which exert a wide general influence through the circulatory system.

The control of the cortex on the diencephalon must be to limit its stimulation by analysing more thoroughly the afferent impulses flowing into the brain, discriminating between those that are harmful enough to call for defensive or offensive reactions and those which are not, and to inhibit the action of the diencephalon.

Functions of the Autonomic Nervous System.

As was said before, the somatic system is concerned with the reactions of the body to its external environment, while the autonomic controls the internal economy of the body. Cannon, in summing up the activity of the autonomic nervous system, states that its ultimate function is to preserve the constancy of the fluid matrix of the body.

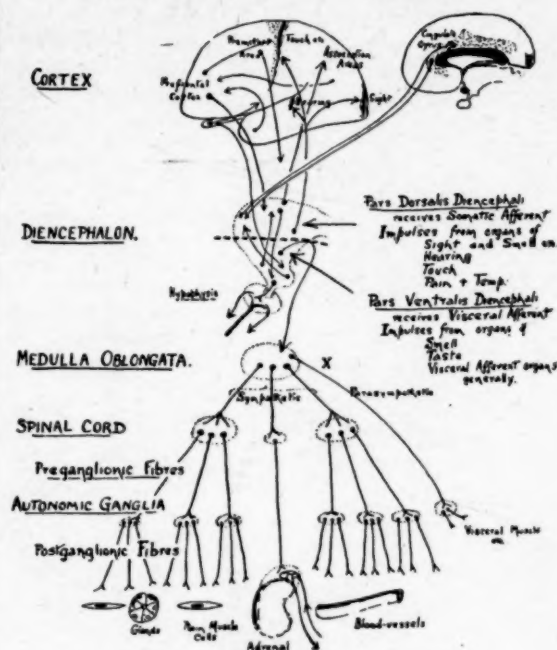


FIGURE IX.
Hierarchy of autonomic centres.

The autonomic system itself is divided into two antagonistic mechanisms, namely, sympathetic and parasympathetic, and this is brought out in Table II.

TABLE II.

Sympathetic.	Parasympathetic
Vaso-constriction Constriction of the pupil Cardio-augmentation Cardio-acceleration Inhibitory to the trachea and bronchi, gut, gastric glands and pancreas Motor to pyloric, ileo-caecal, and internal anal sphincters. Inhibitory to bladder Motor to sphincter of bladder	Vaso-dilatation Dilatation of pupil Cardiac inhibition Motor to trachea, bronchi, gut, gastric glands and pancreas Inhibitory to pyloric, ileo-caecal and internal anal sphincters Motor to bladder Inhibitory to sphincters of bladder

The antagonistic influence of the two systems can be best shown by reference to the following series of diagrams, showing the innervation of some of the important hollow viscera (see Figures X, XI, XII and XIII).

In each case the motor fibres are labelled "M", the vessel fibres "V", and afferent fibres "S".

First consider Figure X, which shows the nervous mechanisms associated with the filling and emptying of the urinary bladder. The nervous

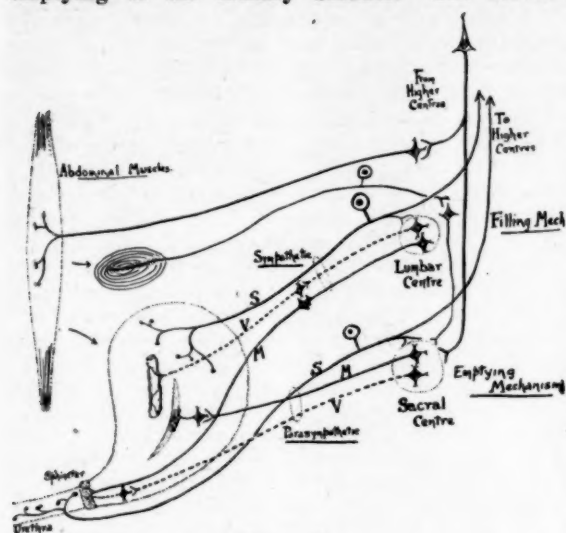


FIGURE X.
Innervation of the urinary bladder.

mechanism for filling has to provide for gradual distension of the bladder and, at the same time, for retention of the urine. This mechanism is innervated by the sympathetic, which, physiologically, is

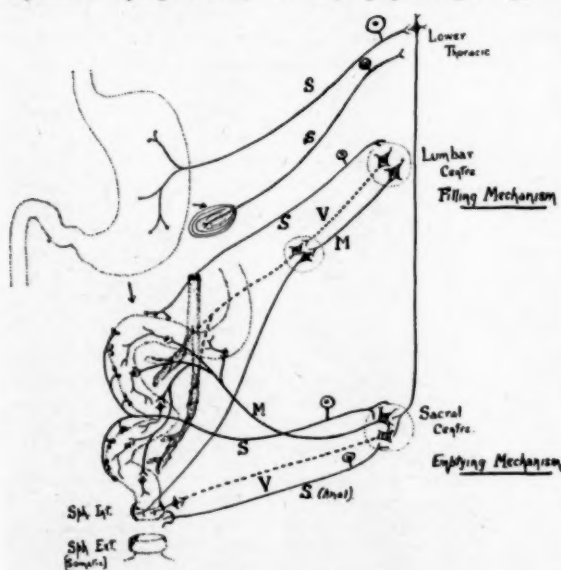


FIGURE XI.
Innervation of rectum.

inhibitory to the bladder wall and motor to the sphincter. Retention is due to the contraction of the sphincter, but the gradual distension of the bladder takes place somewhat as follows.

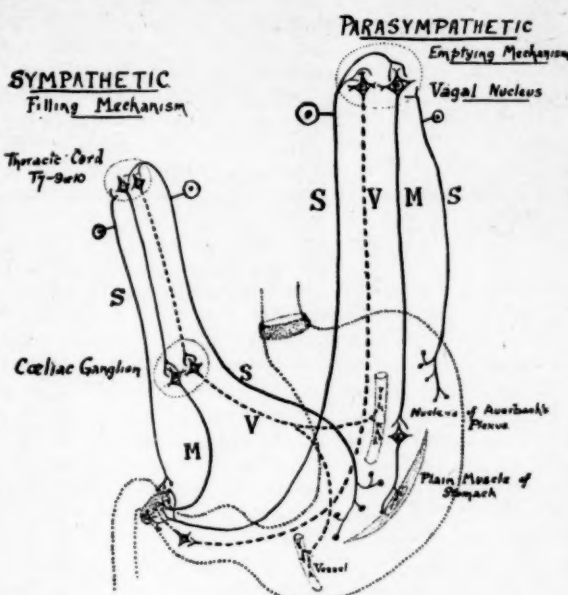


FIGURE XII.
Innervation of the stomach.

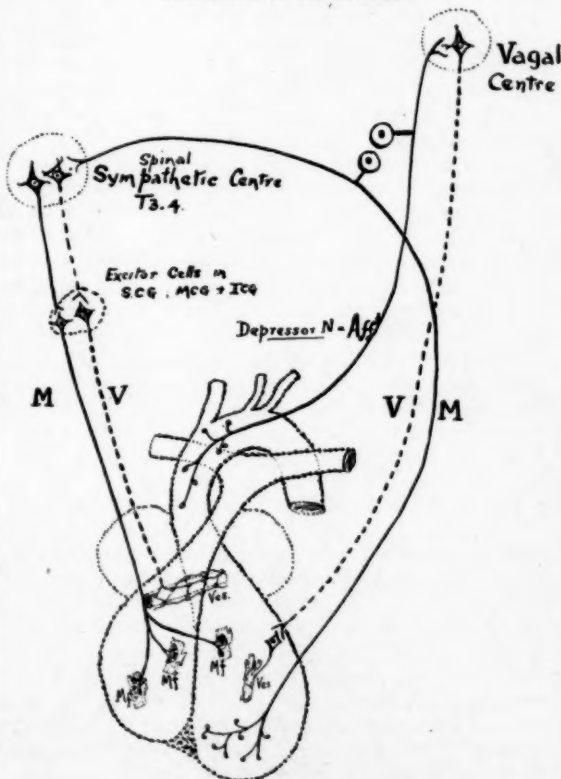


FIGURE XIII.
Innervation of heart.

As the urine is forced into the bladder by the peristalsis in the ureters, the intravesical pressure tends to increase. This stimulates the afferent

endings so that reflex inhibition through the sympathetic centre causes just sufficient "relaxation" to enable the extra urine to be accommodated. The "relaxation" is due to elongation of the muscle cells, but the muscle cells are continually under the influence of the parasympathetic, which maintains their tonus. When the bladder is empty the muscle cells are shorter and stouter, and when the bladder is distended they are longer and thinner; but their tonus remains such that, during filling, the intravesical pressure tends to remain more or less constant. In other words, during filling the muscle cells of the bladder wall continually elongate (sympathetic influence), but remain in a continual state of tonus (parasympathetic influence).

The emptying mechanism of the bladder is provided by the parasympathetic which, physiologically, is motor to the bladder and inhibitory to the sphincter. Stimulation of the sacral centre therefore causes contraction of the bladder wall and relaxation of the sphincter, with consequent emptying of the bladder.

In the case of the bladder there is a point of special interest. The urethral epithelium is particularly well supplied with sensory nerve endings, which are very sensitive to the presence of urine, so that when the intravesical pressure is sufficient to cause a drop of urine to pass into the urethra, the call to micturition is greatly intensified, and, in some cases, irresistible. In initiating the act of micturition, contraction of the abdominal muscles and of the diaphragm increases the intravesical pressure. This causes a little urine to pass through the sphincter into the urethra, with consequent reflex stimulation of the sacral parasympathetic centre facilitating the emptying of the bladder. In the case of the bladder we therefore really have to consider three mechanisms: filling, initiating the act of micturition, and, finally, emptying the bladder.

Figure XI is a diagram of the innervation of the rectum, and the mechanisms are essentially similar to those of the urinary bladder. There is the sympathetically controlled filling mechanism and the parasympathetically controlled emptying mechanism. Corresponding to a sensitive urethral epithelium we have a very sensitive anal epithelium, stimulation of which tends to facilitate defaecation through reflex stimulation of the sacral centre. In this figure the accessory abdominal muscular mechanism is similar to that for micturition and has been omitted. The diagram, however, shows how the increased intragastric and intraabdominal pressure occurring after a meal may reflexly tend to stimulate the sacral centre, with a consequent call to defaecation. Just how much is reflex and how much due to general increased peristalsis from the presence of food in the alimentary canal is difficult to say.

Figure XII shows the innervation conditions in the stomach. It is seen that the vagus (parasympathetic) exerts its influence on the active contraction of the stomach wall through Auerbach's myenteric plexus, while the sympathetic exerts an inhibitory

influence on the stomach wall and is motor to the pyloric sphincter. Stimulation of the parasympathetic produces conditions which tend towards emptying, while the sympathetic acts as it does in the bladder as a filling mechanism.

It will be seen that in all the above cases the sympathetic provides the innervation of the filling mechanism, and the parasympathetic the emptying mechanism of the viscus concerned.

The case of the heart is special (see Figure XIII). The heart must be regarded as consisting of two parts: (i) the cardiac musculature, which forms the wall of a part of the circulatory system and the motor activity of which is therefore controlled by the sympathetic as elsewhere; and (ii) the smaller coronary vessels. These latter are innervated by the parasympathetic, an antagonistic mechanism being thus provided. Just how inhibition is produced is not thoroughly understood, but the histological facts seem to make it certain that here, as in other viscera, the musculature of the organ itself is supplied by one set of autonomic nerves (sympathetic), while the smaller (muscular) coronary vessels are supplied by the other set of autonomic nerves (parasympathetic), the influence of the one being antagonistic to that of the other.

Chemical Factors.

It appears from recent biochemical research that the antagonistic effects of the two sets of nerves in effector mechanisms are due to the liberation, at the nerve endings, of different chemical substances. For example, all efferent fibres which leave the central nervous system, namely, somatic motor and autonomic pre-ganglionic fibres, are said to liberate at their endings a substance akin to acetyl-cholin; and so these fibres are referred to as being cholinergic (Dale). This is significant, because it has been shown that when the somatic muscles are paralysed following the destruction of the somatic efferent (cholinergic) neurones which supply them, they can be brought under the control of the central nervous system again through sympathetic connector cells (also cholinergic) by suturing the central cut end of the sympathetic chain to the peripheral stump of the degenerating somatic nerve (Ballance). Under these conditions the pre-ganglionic fibres of the sympathetic connector cells grow down the somatic path and establish relations with the striated muscle fibres, and these are thus brought again under the influence of the cortex.

The excitor cells of the sympathetic autonomic system, on the other hand, are believed to liberate a substance (sympathin) which is allied to adrenaline. (Figure XIV). The post-ganglionic sympathetic fibres are therefore called adrenergic. It is possible that further chemical research will demonstrate that the parasympathetic fibres also liberate a chemical substance, which is antagonistic in action to that of sympathin, and that in visceral effectors the antagonistic effects of the sympathetic and parasympathetic are really due to the liberation of two different chemical substances.

Another interesting case of this has recently been put forward by Lewis in his work on the cutaneous vessels. He has shown that stimulation of cutaneous sensory nerves can cause local vaso-dilatation. This is due to the passage of impulse along the collaterals

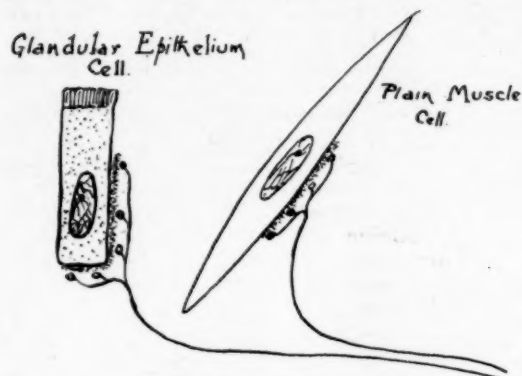
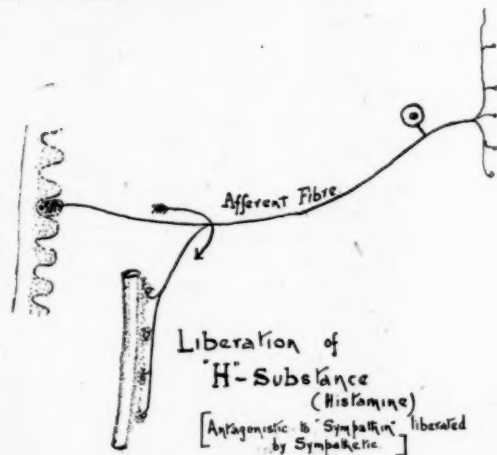


FIGURE XIV.

Pericellular endings of autonomic sympathetic fibres; "sympathin" liberated at the endings.

of the sensory nerves which end in relation to the underlying vessels (see Figure XV). These impulses cause the liberation of a substance akin to histamine, and therefore called by Lewis H-substance. This is antagonistic in influence to sympathin liberated by the sympathetic vasomotor nerves; hence the dilatation.

FIGURE XV.
The axone reflex.

Sympathetic Stimuli.

A matter of clinical interest is in connexion with adequate stimuli for sympathetic stimulation. The most powerful stimuli are hæmorrhage, cold, anoxæmia and muscular work. Hæmorrhage leads to increased coagulability of the blood and peripheral vaso-constriction. Cold produces peripheral vaso-constriction, erection of hairs, and increased metabolism by outpouring of adrenaline. Associated with anoxæmia we have tachycardia, rise in blood

pressure and contraction of the spleen, the significance of which is obvious. Muscular work leads to a rise in blood sugar liberated from storage, dilatation of the bronchioles to admit more oxygen to the lungs, and vaso-dilatation and sweating to regulate the body temperature.

These functions are constantly being exhibited, and when adequately antagonized by the parasympathetic are responsible for regulating visceral activity and maintaining the fluid equilibrium, body temperature and reaction of the blood.

Hypoglycæmic Reaction.

Another interesting sympathetic effect is that associated with low concentration of sugar in the blood. When the blood sugar falls below 70 milligrammes *per centum* we get what is called the hypoglycæmic reaction. The pupils dilate, the skin becomes pale, the blood pressure rises, sweating occurs, and adrenaline secretion is increased, which leads to the conversion of glycogen to sugar and eventual restoration of the sugar level in the blood.

Emergency Sympathetic Reaction.

The emergency sympathetic reaction is akin to the hypoglycæmic reaction. It is designed to prepare the animal for fight or flight and is an expression of the whole sympathetic system at the height of its power, being produced by emotional and psychic stimuli which involve reactions in consciousness and are associated with the activity of the cerebral cortex (Gask and Ross). The pupils dilate so that the visual field is increased. Goose-skin appears. This is a vestigial remnant of a mechanism whereby a hair-covered animal is able to make itself appear larger and more formidable. There is vaso-constriction, the blood pressure rises and the heart beat is accelerated. These increase cardiac efficiency. The spleen contracts and causes an increase in the circulating corpuscles. Glucose is liberated from the liver to supply the needs of the muscles. The bronchi are dilated to increase the oxygen supply. Finally adrenaline is secreted by the suprarenals, and this enhances all the effects already produced by sympathetic stimulation.

Surgery and the Autonomic Nervous System.

It is only in the last decade that the attention of the surgeon has to any great extent been directed towards the autonomic nervous system as a field for surgery. It is true that the early scientific work of Gaskell and Langley was soon followed by surgical experiments, but without any conspicuous success. William Alexander, of Liverpool, for example, removed the superior cervical ganglion in cases of epilepsy as long ago as 1889. As far as I have been able to find out, Alexander was the first surgeon to perform an operation on the autonomic system. In 1899, Jaboulay, of Lyons, stripped off the outer coat of the femoral artery for perforating ulcer of the foot; and again, in 1916, Jonnesco excised the upper part of the thoracic sympathetic chain for *angina pectoris*. Very little, however, was

done in the way of surgery on the autonomic nervous system until 1924, when Royle and Hunter, of Sydney, applied their researches into the sympathetic system in animals to the surgical treatment of spastic states in man. Their work attracted a great deal of attention and led to a great amount of research, and now, at the present day, the operation of sympathectomy is a well established surgical procedure.¹

CONCLUSION.

From what has been said this evening you have gained some idea, I hope, of the recent progress which has been made into the investigation of the autonomic nervous system, and an attempt has been made to stimulate your interest in the clinical application of the new knowledge with which we have been provided. Especial honour is due to the genius of the two Australians who initiated this world-wide activity into the surgical possibilities of this system, and it gives me great pleasure to pay some small tribute to them. Finally, I should like to take this opportunity of deploring the catastrophe which removed one of them, the late Professor John Irvine Hunter, at such an early age from his earthly fields of activity.

BIBLIOGRAPHY.

In addition to the recent editions of standard text books of anatomy and physiology, the following authors were consulted: Charles Ballance: "The Conduct and Fate of the Peripheral Segment of a Divided Nerve, etc.", 1934.

J. Beattie: *Canada Medical Association Journal*, Volume XXVI, page 466. (This contains further references to recent literature on the hypothalamus.)

W. E. Le Gros Clark: "An Experimental Study of Thalamic Connections in the Rat", *Philosophic Transactions of the Royal Society*, London, Series B, Volume CCXXII, page 1; "The Structure and Connections of the Thalamus", *Brain*, Volume LV, page 406.

W. E. Le Gros Clark and R. H. Boggan: "On the Connections of the Anterior Nucleus of the Thalamus", *Journal of Anatomy*, Volume LXVII, 1933; "On the Connections of the Medial Cell Groups of the Thalamus", *Brain*, Volume LVI, 1933, page 83.

J. F. Fulton, M. A. Kennard and H. R. Viets: "The Syndrome of the Premotor Cortex in Man", *Brain*, Volume LVII, March, 1934.

Bibliographies will be found in the following:

Gask and Ross: "Surgery of the Sympathetic", 1934.

Langdon Brown: "The Sympathetic Nervous System in Disease", "Oxford Medical Publications", Second Edition, 1923.

Reports of Cases.

RUPTURE OF THE UTERUS.

By H. A. RIDLER, M.B., Ch.M. (Sydney), F.R.A.C.S.,
Honorary Surgeon, Royal Hospital for Women;
Tutor in Obstetrics, University of Sydney.

Rupture of the Uterus and Bladder.

Mrs. A.B., aged thirty-four years, had three previous confinements and one miscarriage. At the first confinement, eight and a half years ago, she had a normal birth, resulting in a 2.0 kilogram (four and a half pound) infant; after this she had a miscarriage. At the second confinement, six and a half years ago, she was delivered naturally of a male infant weighing 2.9 kilograms (six and a half pounds). At the third confinement, fourteen months ago, she was delivered with instruments of a 3.6 kilogram

(eight pound) baby, which lived three hours. The duration of the labour was twelve hours. During this puerperium she had a lot of bleeding and was much weaker, taking a long time to regain her usual health; after the other two she was quite well.

At the time of her final confinement she was two weeks over her estimated date. After about nine hours' labour she felt (to use her own words) "something give way, and had a terrible pain and felt very ill". She was given a sedative. The regular uterine contractions ceased. I saw her ten hours later and diagnosed a ruptured uterus, the beginning of the rupture being felt in the cervix from the external os upwards. She refused to be operated on until she had seen her husband; this caused a delay of several hours.

On catheterization the bladder also was found to be ruptured.

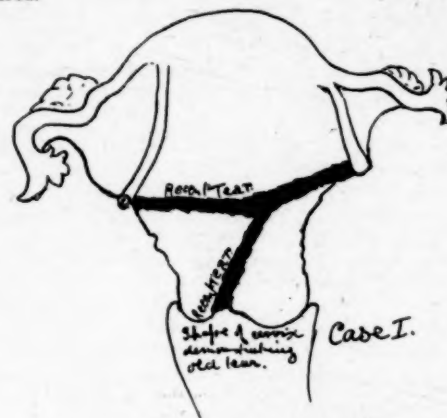


Diagram showing rupture of the uterus.

On opening the abdomen, the fœtus, which weighed 3.0 kilograms (six pounds fourteen ounces), and the placenta were in the peritoneal cavity. The tear was T-shaped. The tail or bottom piece started on the right side of the cervix, running upwards anteriorly and to the left, and involved the posterior wall of the urinary bladder. The top or cross piece extended from one uterine artery to the other, just short of both, being in the lower uterine segment, just below its junction with the upper (see diagram). The uterus was easily removed by clamping both uterine arteries at the ends of the tear and dividing the uterus posteriorly. The body of the uterus being out of the way, one could see down into the vagina to the lower end of the bladder tear; the bladder was sutured, the stump of the uterus stitched across and, finally, the peritoneum as in a subtotal hysterectomy. The abdomen was closed and a self-retaining catheter was put into the bladder through the urethra.

The patient recovered, but had a small vesico-cervical fistula; this was repaired fourteen weeks later.

My explanation for this rupture of both bladder and uterus is that there must have been an extensive tear of the cervix, probably extending upwards into the lower uterine segment, at a previous confinement, and when this tear healed the bladder became adherent to this scar tissue.

From the patient's history, this tearing took place at her third confinement; also from the history the rupture in the final confinement was not due to disproportion, but to obstruction by scar tissue in the cervix, which tends to tear rather than stretch, and when the tearing once started would continue till the uterine contractions ceased. This would be when the fœtus was expelled from the uterus; in this instance into the peritoneal cavity.

Rupture of Previously Perforated Uterus.

Mrs. C.D., aged thirty-four years, had two children, four and three years old, followed by two miscarriages, the last ten months ago, for which her uterus was curetted; this was her only operation.

¹ The last part of the lecture dealt with sympathectomy for disorders of the circulation and the methods employed to enable the surgeon to forecast the benefit which is likely to arise from the operation; but as the facts are all set out *in extenso* in the admirable manual by Gask and Ross, there is no need to repeat them here.

She was pregnant again, being nearly six months. At about six o'clock one evening she began to get pains and slight bleeding. About eight hours later I was asked to see her, as she had suddenly collapsed, having a very rapid pulse, subnormal temperature, rigid and tender abdomen, and general pallor, with only a little vaginal hæmorrhage.

On vaginal examination the cervix would admit one finger only, but nothing could be felt in the uterus.

She was prepared for immediate operation, and under the anæsthetic a depression could be felt in the top of the fundus, as the patient's abdominal wall was very thin. This suggested a ruptured uterus.

On opening the abdomen, the rupture in the top of the fundus resembled the ruptured scar of a pregnant uterus following a previous Cæsarean section, but no Cæsarean section had been performed on this patient; as previously stated, her only operation was a curettage of the uterus.

The placenta, attached to a complete bag of unruptured membranes containing the fœtus and liquor amnii, was found in the peritoneal cavity.

This rupture in the fundus was easily and quickly repaired and a blood transfusion given, but the patient died on the fourth day from general peritonitis.

For such a condition to occur the uterus must have been extensively perforated when curettage was performed ten months previously.

Comment.

When a patient has had a rupture of the uterus, Cæsarean section should be done for future pregnancies. An extensive perforation of the uterus is in the same class as a rupture of the uterus.

ENDEMIC TYPHUS IN NEW GUINEA.

By CARL E. M. GUNTHER, M.B., B.S., D.T.M. (Sydney),
Bulolo, Territory of New Guinea.

THE details of the following cases may be of value in assessing the probable nature of the variety of endemic typhus which has recently assumed some importance on the goldfields of the Morobe district of the mandated Territory of New Guinea.

These are the only cases which the writer has handled personally; and while one hesitates to draw conclusions from two cases only, yet in view of the many features which they have in common, one is perhaps justified in assuming certain points as a basis for future investigations.

Ætiology.

Both men are employed by Bulolo Gold Dredging, Limited, as oilers on number 3 dredge, operating on the Bulwa leases. For the dredges to operate, the ground must be cleared thoroughly. The crews of number 3 dredge reach their work by a track which traverses the freshly felled bush. The crews of the other dredges are transported by truck to the site of operation. Neither of these men is in the habit of spending leisure hours wandering about the bush. Hence the only likely place where the infection could have been acquired is this area of freshly felled bush.

The tempting hypothesis that infection follows the bite of a larval trombicula, known throughout the territory as the "bush-mokka", has yet to be proved; but to the writer it appears to be most reasonable.

The main features of the two cases are set out in Table I.

Serum Reactions.

Unfortunately the first case was a surprise, and emulsions of *Bacillus proteus* were not available until some months later.

Technique.—Serum was collected from venous blood in the usual way and a series of dilutions from 1 in 10 to 1 in 160 was made. Two series of tubes were put up for each serum, one with *Bacillus proteus* Warsaw, the other with *Bacillus proteus* Kingsbury. The emulsions were Commonwealth Serum Laboratory products; the same fine-

TABLE I.
Comparison of Cases I and II.

	Case I.	Case II.
Date of onset..	August 19, 1934.	December 28, 1934.
Prodromal symptoms..	Nil.	Nil.
Onset ..	Sudden. Mild rigors. Temperature around 38.9° C. (102° F.). Pulse rate around 76 per minute.	Sudden. No rigors. Temperature around 38.9° C. (102° F.). Pulse rate around 100 per minute.
Fever and pulse rate ..	Temperature averaged 39.1° C. (102.5° F.) at 2 p.m. and 38.3° C. (101° F.) at 6 a.m. Pulse rate averaged 76 per minute.	Temperature averaged 39.4° C. (103° F.) at 6 p.m. and 38.6° C. (101.5° F.) at 10 a.m. Pulse rate averaged 100 per minute.
Duration of fever	Lysis commenced on thirteenth day. Temperature normal on seventeenth day.	Lysis commenced on tenth day. Temperature normal on fourteenth day.
Rash ..	Appeared on trunk and limbs, not on face. Irregular, rose-coloured spots, averaging seven millimetres in diameter. Brighter and more closely set over areas of pressure. Disappeared on pressure, returning at once. Appeared on ninth day. Faded by eleventh day. Gone on twelfth day.	Appeared on seventh day. Faded on eighth and ninth days. Gone on tenth day.
Eschar ..	Right shin. Typical. Central black slough four millimetres in diameter; red areola fourteen millimetres in diameter. No pain, tenderness or itching. First noticed on twelfth day. Well developed. Excised on fourteenth day. Healed slowly.	Left calf. Atypical. Dusky area twelve millimetres in diameter; later, central area of desquamation three millimetres in diameter. No pain, tenderness or itching. First noticed on eighth day. Not excised. Disappeared by eighteenth day.
Other symptoms	Headache, insomnia and nightmares (severe). Obstinate constipation. Anorexia. Tongue dry and furred.	Headache, insomnia and nightmares (mild). Mild constipation. Anorexia. Tongue dry and furred.

drawn Pasteur pipette with rubber teat and square-cut tip was used for all measurements, being thoroughly washed with saline solution after each time of using.

Five drops of the successive dilutions of serum and five drops of emulsion were placed in each tube, giving final dilutions of serum of 1 in 20 to 1 in 320. Controls were put up for each series, containing five drops of saline solution and five drops of emulsion.

The tubes were then placed in a water bath at 37° C. for two hours.

As a matter of interest, serum from another man who had had tropical typhus in Malaya three and a half years previously was also tested (Case III).

Widal tests were performed in Cases I and II, with positive results at low titre—Case I in a serum dilution of 1 in 160, and Case II of 1 in 320 (Table II). These results were accounted for by anti-typhoid inoculations within the previous twelve months.

Conclusions.

As far as can be determined from only two cases, it seems reasonable to make some tentative remarks as a basis for future comparison. The fact that the two cases agree in general to a remarkable extent supports this contention.

1. The cases correspond clinically and serologically with the widespread class of fevers grouped as endemic typhus.

2. The cases exhibited certain similar features: fever lasting about two weeks, ending by lysis, with a relatively low pulse rate; a typical rash on trunk and limbs,

TABLE II.
Results of Testing with various Dilutions of Emulsions of *Bacillus proteus*.

	Titre.					Control.
	1/20	1/40	1/80	1/160	1/320	
Case I, five months after—						
<i>K</i>	+	—	—	—	—	—
<i>W</i>	—	—	—	—	—	—
Case II, on eighth day—						
<i>K</i>	+	+	+	—	—	—
<i>W</i>	—	—	—	—	—	—
Case II, on sixteenth day—						
<i>K</i>	+	+	+	+	—	—
<i>W</i>	—	—	—	—	—	—
Case III, three and a half years after—						
<i>K</i>	—	—	—	—	—	—
<i>W</i>	—	—	—	—	—	—

NOTE.—*K* = *Bacillus proteus* Kingsbury; *W* = *Bacillus proteus* Warsaw.

appearing early in the second week and fading in three days; constipation; headache, insomnia and nightmares; anorexia; an eschar at a typical site.

3. The cases agglutinated *Bacillus proteus* Kingsbury, but not *Bacillus proteus* Warsaw.

Reviews.

THREE PHILOSOPHERS.

W. R. AYKROYD, in the book bearing the title "Three Philosophers", offers us a fine sample of the art of writing biography. He deals in an attractive manner with the lives and doings of three famous chemists—Lavoisier, Priestley and the Honourable Henry Cavendish—and shows how the Frenchman laid the foundations of modern chemistry in virtue of the fact that he made use of the other two. Priestley met Lavoisier but once, when, owing to the Englishman's halting French, their conversation consisted in nothing but a laboured discussion on scientific topics. Cavendish, the wealthy hermit, Lavoisier never saw. But Priestley's discovery of oxygen, together with Cavendish's researches into the composition of water, and on nitric acid, lit the fire which led Lavoisier to overthrow the old phlogiston theory of Stahl, to explain the true nature of combustion, to lay the foundations of quantitative chemistry by the proper use of the balance, to introduce a rational system of classification and nomenclature, and to prosecute the earliest investigations into human metabolism. There is no manufacturing chemist of today assailing the market with the latest cure for the ills of mankind but owes something to these three.

Yet how amazingly different in upbringing, fortunes and habits of thought the three men were: Priestley, a non-conformist minister, hounded from place to place, even suffering the loss of his house and church by fire at the hands of a yelling mob, but through the years carrying on with meagre apparatus his chemical experiments; Cavendish, the offspring of dukes, fabulously wealthy, yet a sour recluse, going nowhere except to the meetings of the Royal Society, writing huge cheques to get rid of chance callers, and snarling at the timid servant who dared to approach his death bed; and Lavoisier, the handsome young man from the ranks of the upper *bourgeoisie*, wealthy, possessed of a pretty and clever wife who loved him and assisted in his laboratory work during his whole working life.

Lavoisier is the central figure of Mr. Aykroyd's tripartite drama. And so it should be, for, though he hated romance, airs and graces, and artists (for he lived in the cold, clear-cut atmosphere of science), he is yet a romantic figure, in spite of himself. Let us in a few words call up the picture of this great Frenchman as Aykroyd sees him.

Lavoisier was always the alert, aloof scientist. He bent himself, in an icy calm, to any problem before him. All things concerning human welfare were fish to his net. He tried to improve the water supply of Paris, in days

when Paris was an evil-smelling warren of narrow streets, and twenty thousand men carted water to the houses of such as could buy it. He advocated reform in hospital and prison construction; he invented a mechanical arm-chair; he inquired into the nutritive properties of gelatine soup; he developed a plan for the better lighting of the streets of the town, where villainies of all sorts were perpetrated under cover of darkness. Such was the man, with a many-sided mind, only second, it may well seem, to that of Leonardo da Vinci, who had to meet death by the guillotine.

The guillotine? Yes, for, although Lavoisier was free-handed to those in need, there was a certain "grabbiness" about him—he loved to make money. It was this trait in his character which impelled him to buy a place in the *Ferme Générale*, the company which for years had purchased the right, in return for the payment of a lump sum to the State, to collect and enjoy all moneys paid as taxes by the groaning people of France. The company collected dues on tobacco, on the salt gabelle, on alcoholic liquors, on a thousand and one things, and levied imposts at the gates of all French towns. Even today tax-collectors cannot expect to be the darlings of the people; in eighteenth-century France, when all but aristocrats raged under the exactions of a privileged few, when ragged peasants were forced to surrender half their earnings to a private company, there could be but one end to it all: the revolution, with all its brutalities, raged across the country. Lavoisier, with a round three score of his fellow *fermiers-généralux*, knelt with cropped hair beneath the descending knife. "Ah," said a bystander, "you cut off his head in a second; it will take a hundred years to grow another like it." And within a few weeks, Robespierre, the arch-butcher Fouquier-Tinville, and the chemist's judge, the bloody-minded Coffinhal, had expiated their own orgy of mass murder upon the scaffold. In 1796, in August of that year, the *Lycée des Arts* honoured the memory of Lavoisier. At the portal, hung a notice: "*A l'immortel Lavoisier*." Inside, among the chandeliers and cypress branches, was a tomb bearing a statue of liberty. The choir sang panegyrics and dirges; and then appeared a bust of the dead man, crowned with a laurel wreath. But it was too late for singing and wreaths; the murder, the conspiracies that seem to be part of a new-found democracy, had wiped out one of the noblest sons of France.

Notes on Books, Current Journals and New Appliances.

AN AUSTRALIAN MEDICAL DIRECTORY.

THE "Medical Directory for Australia", published by Errol G. Knox, will be an extremely useful book of reference both in Australia and overseas.¹ For many years the need has been felt for the publication of a book of this kind. Mr. Knox is to be congratulated on his enterprise. Before publication he sought the approval of the Federal Council of the British Medical Association in Australia; this approval was given.

The main portion of the book is an alphabetical directory of Australian practitioners. The qualifications, hospital appointments, offices held in and membership of the British Medical Association and other medical and scientific societies, war service, honours and titles are stated, together with the names of publications. The alphabetical directory is followed by a gazetteer in which practitioners are enumerated according to their place of residence. Then follows information about the Australian medical boards, the Faculties of Medicine in the universities of Australia, the British Medical Association in Australia, and the Royal Australasian College of Surgeons.

The book has been well printed and the binding is attractive. We hope that Mr. Knox's venture will meet with the success that it deserves and that subsequent editions will be published from time to time.

¹ "Medical Directory for Australia, 1935", by G. H. Knox; 1935. Sydney: Errol G. Knox. Demy 8vo., pp. 769. Price: 51s. net.

¹ "Three Philosophers (Lavoisier, Priestley and Cavendish)", by W. R. Aykroyd; 1935. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 227. Price: 10s. 6d. net.

The Medical Journal of Australia

SATURDAY, JUNE 29, 1935.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

A TWENTY-FIRST BIRTHDAY.

WITH this issue THE MEDICAL JOURNAL OF AUSTRALIA completes its twenty-first year—born on July 4, 1914, it has attained its majority. The anniversary is a matter for satisfaction and congratulation; it also carries with it a challenge for the future, a challenge to every member of the British Medical Association in Australia.

When the journal was established in 1914 it was intended to act as the official organ of the Branches of the British Medical Association in Australia. This was, as most of our readers know, one of the results of the formation of the Federal Committee. It may fairly be claimed that throughout the twenty-one years of its existence the journal has upheld the ethical standard of the British Medical Association; it is also a cause of great satisfaction to those controlling the journal that it has received the whole-hearted support of the several Branches. Not that there have been no differences of opinion; differences have occurred, but they have invariably been adjusted in a spirit of understanding and with a desire for the common good. During the past

twenty-one years Australia has passed through economic upheavals. War broke out just one month after the establishment of the journal. During the years of the War financial worries, such as those connected with the price of paper, caused much anxiety, but with care and economy the storm was weathered. During the years of comparative plenty that followed, the Australasian Medical Publishing Company, Limited, which controls the journal, expanded its activities, erected The Printing House and equipped it with a complete plant. It engaged a skilled staff of type-setters, compositors, readers and machinists, who have given of their best to the undertaking. The financial depression was the second crisis that had to be faced. From this the company has emerged successfully, and it now finds itself faced with an enormous amount of work. The need for more machinery has recently been experienced, and to meet this need the Company's building has been extended, so that it now occupies the whole of the land belonging to it. A picture of the completed building appears on another page in this issue. The Branches are to be congratulated on possessing, through the medium of the Australasian Medical Publishing Company, Limited, a complete scientific printing establishment that is large enough to meet the needs of the Association for several years to come.

The number of journals published in the modern medical world is enormous; "Index Medicus" deals every year with several hundreds of journals. Many medical journals are ephemeral, and doubtless quite a few are superfluous. A well known Australian clinical teacher has declared that two-thirds of the material published in medical journals is rubbish. Herein lies the challenge to Australian members of the British Medical Association. The functions of THE MEDICAL JOURNAL OF AUSTRALIA, as set out in the leading article in the first issue, are two. The first is the recording of scientific medicine; the second is that it shall assist in rendering the practice of medicine in all its branches of the greatest benefit to the people of Australia. A moment's thought will show that these two cover most of the activities of the journal. In regard to the first, we

would point out that many contributions to scientific knowledge are made in the Commonwealth. The account of work done in Australia should, whenever possible, be published in Australia. Any important Australian work published in Australia will direct to the Commonwealth the eyes of workers in other parts of the world, and incidentally will enhance the value of the journal in which it is published. Australian scientific journals have large exchange lists with journals of other countries. The scientific work of other countries is brought to the notice of readers of this journal, as far as possible, in the editorial columns and in the section devoted to abstracts from current medical literature. In recording the proceedings of the Branches of the British Medical Association and in acting as a medium of post-graduate instruction, this journal is helping to render the practice of medicine of benefit to the people of the Commonwealth. Its effectiveness in this regard depends on the value of the material offered for publication. It is therefore clear that the status of THE MEDICAL JOURNAL OF AUSTRALIA depends to a very large extent on the efforts of individual practitioners—no editorial staff can, unaided, lift a journal from the ruck and keep it in the front rank among those worthy of the epithet scientific.

Current Comment.

AGRANULOCYTOSIS.

No investigator has so far succeeded in laying the ghost of the usually fatal disease variously called agranulocytosis, agranulocytic angina, and malignant neutropenia. References to it have previously appeared in the columns of this journal¹ and in those of British publications,² but the subject has aspects of such importance that no apology is needed for its reintroduction. A few years ago so little was known of the disease that although it had been noted, named and described by certain German observers, and although Lovett³ and Skiles⁴ have prosecuted important researches into its cause and character, it yet receives but cursory notice in any

text book and is, probably, but little considered by the ordinary practitioner as a clinical entity.

The reason is obvious; agranulocytosis, previously unsuspected, may first manifest its presence by the appearance of ulcers, spreading like a bush fire upon the tonsils, gums, larynx, tongue and pharyngeal walls, and even upon the genitalia. The patients (many of them middle-aged women) usually die within a few days. It is easy to understand how, during such dramatic episodes, errors in diagnosis may constantly arise, and how such patients may be judged to have suffered from any one of a category of disorders—aplastic anæmia, acute leucæmia, Vincent's angina, influenza, or diphtheria. In this disease the responsibility for diagnosis rests upon the hæmatologist, and the blood picture may be, and usually is, altered long before the ulcerative lesions are seen. The white cells may fall to fantastically low levels (even to 100 to 200 per cubic millimetre). The polymorphonuclear cells of all kinds may be said to vanish, leaving a high proportion of lymphocytes. The red cells usually remain unaffected, though in rare instances an anæmia of macrocytic type may be discovered.

In the light of recent experiences of this condition, J. F. Wilkinson¹ defines agranulocytosis as a recently recognized disorder manifesting very characteristic blood changes and commonly proceeding, so far as is at present known, to a fatal ending. The onset is dramatically acute; the patient, overcome by malaise, headache and lassitude—by severe symptoms which seem to bear no relation to the somewhat trivial bacteriological findings so far recorded—is soon further prostrated by the appearance of spreading, often necrotic ulcers on the tongue, fauces, pharynx, gums and even cutaneous surfaces. Happily, occasional remissions occur; but only too often the disease kills within a few days. As stated, the bacteriological examinations have invariably revealed the presence of germs of low toxicity, a fact perhaps explained on the assumption that the buccal and other lesions are a secondary complication to the disease proper. Colour is lent to this view by Wilkinson's demonstration of the characteristic blood picture many months before any morbid changes appear about the mouth. A further strange feature, the author states, is the existence of rare cases of chronic agranulocytosis in which the granular leucocytes may remain of the order of 10% to 30% for long periods of time.

The aetiology of this strange disease is an elusive problem. It is sure, however, that a single dose of such a drug as pyrimidon will, in a susceptible patient, cause a perilous reduction in the granulocyte content of the blood. This effect has been produced by so small a quantity of pyrimidon as five to ten grains. Other preparations belonging to the "benzamine" group are also under suspicion. This aspect has been referred to in this journal. Since the mortality rate in the disease is assessed as between 60% to 90% it must be confessed that

¹ THE MEDICAL JOURNAL OF AUSTRALIA, March 31, 1935.

² The Lancet, January 5, 1935.

³ The Journal of the American Medical Association, Volume LXXXIII, 1924, page 1498.

⁴ The Journal of the American Medical Association, Volume LXXXIV, 1925, pages 364 and 1415.

⁵ The Practitioner, March, 1935.

universally efficient treatment, so far, has not been instituted. Nevertheless, Wilkinson now reports hopeful results from the use of a mixture of the sodium salts of pentose nucleotides ("pentnucleotide") administered parenterally. Observation of blood-counts performed daily have indicated progressive increases in the numbers of white cells, and the treatment has in a few instances had a favourable influence upon the oral and other lesions.

A well known Australian physician who has devoted time and thought to the subject of agranulocytosis has had difficulty in collecting more than a small quantity of relevant clinical material. It would appear that the largest hospitals can, as yet, produce from their records very few data bearing upon the disease. One may hazard the suggestion, however, that there have been in the past cases of mistaken diagnosis in this regard. It is at least an intelligent guess that some patients unsuccessfully treated for some infective condition of the upper air-passages, or for aplastic anaemia, leuchæmia or even Hodgkin's disease, have died, in reality, from agranulocytosis.

THE ÆTIOLOGY OF HYPERTENSION.

It is unlikely that any disease syndrome which affects the more complex and widespread systems of the body is due to a single cause. Even where the morbid process concerns a delimited area the problem of its ætiology is difficult enough, but in the case of the vascular tree it is obvious that there may be many factors at work. Atheroma, using the term to denote only the characteristic degenerative processes attacking the intima of the larger vessels, is certainly due to several causes. Hypertensive vascular disease, which affects a much larger volume of the blood channels, namely, the lesser arteries and arterioles, is still more complicated; but though the riddle is not yet solved, certain aspects of it are becoming clearer. Thus it is established that in some cases of pituitary disease hypertension is common; this refers to those curious cases of pituitary basophilism to which Cushing has drawn attention. At first sight this seemed to offer a ready solution of some of the difficulties surrounding the study of hypertension, but it has been pointed out that autopsy has revealed the presence of basophile adenoma where no signs of the curious Cushing syndrome had been observed during life. Further, infiltration of the basophile cells of the pituitary seems to be a feature of later life apart from morbid change elsewhere; and, finally, the basophile syndrome is usually associated with changes in other ductless glands, especially the suprarenal, leaving us in doubt as to which, if any, is the true culprit. However, stimulated by this idea, attempts have been made to correlate the presence of small adenomata in the basophile part of the pituitary with a history of hypertension during life.

B. A. Gouley describes a case of the Cushing syndrome that is interesting, because the striking

appearance of the patient as observed at autopsy led to a tentative diagnosis of basophilic adenoma, confirmed by histological examination.¹ This patient died after only three days in hospital from a classic capsular hæmorrhage, the study of the blood pressure, the retinae, the blood and the urine establishing the cause as hypertension and *diabetes mellitus*. There can be no doubt that the hirsuties, the hypertension, the obesity, the glycosuria and the demonstrated adenoma of the pituitary, together with adrenal hyperplasia, place this case in the category of the Cushing syndrome. It illustrates how an ordinary routine autopsy in a case of cerebral hæmorrhage might easily miss what was in this case the most important point in the ætiology.

From this we turn to the general question of the relation of the hypertensive disorders to basophilic invasion of the hypophysis. Charles Spark has reviewed the subject from the historical aspect and has also presented the results of a study of the pituitary apparatus of 70 persons who had suffered from essential hypertension, 11 others who had signs of other types of hypertensive disease, and 108 controls.² He points out that the findings of other workers indicate the tendency for basophilic infiltration to occur with advancing age; and with this conclusion he agrees, considering that this particular cellular activity is related to the age of the person, and possibly to some sex factor as well. But, more important still, he finds that there is no connexion apparent between the degree of basophilic invasion of the *pars nervosa* and the presence of hypertension. When groups of similar ages are compared there is no greater amount of this type of pituitary change among those known to suffer from essential hypertension during life. In addition, Spark cannot find evidence for believing that either eclampsia or essential hypertension is due to any hyperactivation of the pituitary apparatus by the basophilic cells. He is prepared to admit that the occurrence of a basophile invasion is due to some form of stimulation; but whether nervous, from hypothalamic centres or not, or hormonal, he is not able to say. It would appear, then, that whatever the rôle of these special cells in causing hypertension, there is not always a direct causal relation.

Attacking the problem from another angle, Bohn, one of the assistants of Volhard, some years ago claimed to have demonstrated pressor substances in the blood of patients suffering from essential hypertension. Confirmation of this important claim has not been obtained by other observers, notably de Wesselow and Griffiths, and further work is now to hand by R. S. Aitken and C. Wilson on the same subject.³ These investigators have also been unable to substantiate Bohn's claim, though they followed his technique of ultra-filtration in the main. They consider, however, that the experimental method he employed has limitations that make it essentially unsuitable for the detection of pressor substances

¹ *Annals of Internal Medicine*, April, 1935.

² *Archives of Pathology*, April, 1935.

³ *Quarterly Journal of Medicine*, April, 1935.

in the amounts regarded as significant by Bohn in his experiments. Whether more sensitive methods would be reliable, or would be likely to yield results of any importance, perhaps further work will indicate. If we ask ourselves where are we left, in the light of these results, we must reply that we are not merely where we were before, because negative knowledge is of great value. At the same time it must be admitted that the riddle of hypertension is not yet solved.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held on March 28, 1935, at the Anatomy Theatre, University of Adelaide, Dr. L. C. E. LINDON, the President, in the chair.

The Autonomic Nervous System.

PROFESSOR H. J. WILKINSON read a paper entitled: "The Autonomic Nervous System" (see page 801).

DR. FRANK BEARE thanked Professor Wilkinson for his most useful paper. He pointed out that the speaker had made little mention of the afferent side of the vegetative nervous system. He wished to remind the meeting that Langley, in his earlier writings, had said that the vegetative nervous system did not contain reflex arcs, as existed in the sensory motor system. This view, however, was not that now held. Anatomical studies had shown that afferent pathways for the vegetative nervous system existed in abundance. The fibres might be either myelinated or unmyelinated and were often mixed up with the fibres of the sensory motor system. These vegetative fibres relayed in the posterior root ganglion and entered the spinal cord by way of the posterior horn. Unfortunately this part of the vegetative system had suffered from neglect at the hands of many histologists. Receptor organs similar to those of the sensory motor system were known to exist in abundance. When these receptors were stimulated, they did not on the whole result in perceptions similar to those resulting from stimulation in the somatic system. Thus pain was little felt in viscera, but an increase in tension might result in extreme discomfort, felt, not in the viscus, but referred to some area on the body surface. Similarly temperature was not felt, except extreme heat, in such regions as the anus, oesophagus and stomach. The above facts led one to the important subject of referred pain. When the receptor organs in a viscus were stimulated strongly, some or all of three mechanisms were set in motion. First, a viscerosensory reflex—pain referred to some segmental area of the somatic system—resulted. One characteristic of this pain was that it was not felt in all the somatic segmental areas, but only a part; thus the pain of appendicular trouble in the early stages was felt only at the umbilical region. With this phenomenon there was an area of hypersensitiveness to painful stimuli applied to the region. Secondly, a visceromotor reflex was manifested by rigidity of certain muscles which were hypersensitive to pressure. And, finally, there was a viscerosecretory reflex, not so commonly met with, and shown by secretion on the part of some gland; for example, he instanced the increased salivation often met with in *angina pectoris*.

Dr. Beare further pointed out that normally the results of stimulation of these afferent nerves from viscera *et cetera* did not enter consciousness. Examples of a similar state existed in the somatic system. Thus no one was normally aware of the rubbing of underwear on the skin.

In states of abnormal mental sensitiveness, however, these vegetative afferent impulses did obtrude into consciousness and the individual became often distressingly aware of heart beat, respiratory movements, and so on. This, of course, was one of the characteristics of an anxiety state and led to wrong interpretations and false ideas of heart disease, lung disease, and so on.

Dr. Beare wished to emphasize what Professor Wilkinson had pointed out about the upper levels of the vegetative nervous system. It had to be realized that this system extended up through the medulla, the mid-brain, the diencephalon, and perhaps finally reached the cortex. As to whether there was direct cortical representation of viscera was a moot point but was at least worthy of consideration.

Modern neurological research had shown that many vegetative centres of great importance existed in the hypothalamic region. It would seem that these centres governed the various metabolic activities of the organism. The vegetative system supplied both the afferent and efferent fibres joining these centres to the various regions of the body concerned with metabolism. Further, it would seem that the endocrine system, through the vegetative nerves, was directly controlled by these centres. In the hypothalamic region centres existed for the control of such important functions as fluid balance, fat and sugar metabolism, heat regulation and many others, in fact all the internal chemico-physical activities of the organism. This hypothalamic region was that lying ventral to the thalamus, and included the *tuber cinereum*. Many nuclei had been defined, and some were actually named and had had functions centred in them. Some of these nuclei acted through the parasympathetic, while others functioned through the sympathetic nerves. This conception had been abundantly substantiated by means of experiment and of clinico-pathological observation and, in Dr. Beare's opinion, would finally solve many as yet puzzling problems in clinical medicine. It would seem that these centres were liable to disturbance from strong emotional impulses, and perhaps this would serve to explain the known "mental" origin of many clinical conditions. He instanced *diabetes mellitus*, arterial hypertension and peptic ulcer.

Finally, Dr. Beare wished to ask Professor Wilkinson some questions. He had referred to certain medullary centres governing the heart beat and respiration. Were these centres anatomical facts or were they physiological hypotheses? Secondly, did vaso-dilator nerves exist similar in nature of action to vaso-constrictor nerves? Dr. Beare had a vague idea of having read some work dealing with the matter. Professor Wilkinson had not mentioned the existence of parasympathetic nerves in the thoracic and lumbar regions. Did these exist? And, if they did exist, were they vaso-dilator in action? And, finally, was any pathway in the spinal cord known to exist by which the sacral parasympathetic travelled downwards?

DR. E. BRITTON JONES referred to the work of John Morley on abdominal pain, in which a view was put forward differing from that of Mackenzie with regard to the mechanism of the so-called referred pain. Morley had described experiments in which he claimed that he was able to show that the area of the referred pain was not fixed in a constant position for all postures of the patient, and put forward the theory that the pain was due to contact of the inflamed area in the viscus with the parietal peritoneum. He said that the afferent nerves involved were thus somatic nerves and that the impulses "radiated" into the subcutaneous nerves coming from the overlying skin area and were thus referred by the brain to this skin area. Dr. Britton Jones asked Professor Wilkinson what effect Morley's work had had on the present-day explanation of abdominal pain.

DR. G. H. BURNELL, in reference to the method used by Gask and Ross, and described by Professor Wilkinson, to enable the surgeon to predict the benefit likely to be derived from sympathectomy for disorders of the circulation, said that he understood that the usual and simplest method was the induction of fever by the protein shock method, such as intravenous injection of triple typhoid vaccine, and the simultaneous taking of the surface tem-

perature of the fingers and toes, and the temperature of the mouth. He wondered whether Gask and Ross's method was supplanting the protein shock method.

Professor Wilkinson, in reply, said that he had deliberately refrained from discussing in detail the afferent side of the autonomic nervous system and its relation to abdominal pain *et cetera*, because the anatomy was not difficult to understand and the discussion of abdominal pain would really require a lecture in itself. It was quite true that the viscera were supplied with endings of afferent neurones, as Dr. Beare stated, and some reference was made to them in his paper. The afferent nerves were of both the medullated and non-medullated variety, and their cell bodies were situated in the posterior root ganglia and in their cranial equivalents, namely, the ganglia on the cranial afferent nerves, such as the vagus. The medullated splanchnic or visceral afferent fibres had been counted, and amounted to only about 2% of the total medullated afferent fibres of the body, namely, 12,000, as against 600,000; but the non-medullated visceral afferents had not been accurately determined. The ratio of somatic to autonomic non-medullated afferents was probably of the same order, so that it could be said that the viscera were not nearly so well supplied with afferent nerves as were the somatic structures.

With regard to the theories of Mackenzie and Morley concerning the mechanism of abdominal pain, and mentioned respectively by Dr. Beare and Dr. Britten Jones. Professor Wilkinson thought that, although there appeared to be difficulties in both, that of Mackenzie was still taught in the schools. There was another explanation, however, which Professor Wilkinson gave to his neurological classes, which seemed to him to be more satisfactory; but as it was getting late and would take some time to expound, it was better to leave it until another opportunity offered. He mentioned that there was a paper in a recent issue of *Brain*, which was written by Charles Bolton in answer to Morley's book on abdominal pain, in which the author described thirty-nine cases of gastric and duodenal ulcer in support of Mackenzie's theory.

In answer to further questions of Dr. Beare, Professor Wilkinson stated that cardiac and respiratory centres in the medulla were regarded as anatomical entities and formed part of the dorsal nucleus of the vagus in the floor of the fourth ventricle. Vaso-dilator nerves had been postulated by physiologists and demonstrated by them, for example, in the *nervi erigentes* (sacral parasympathetic); and certain Japanese investigators claimed to have demonstrated them in spinal nerves from other spinal segments, but this work had not been confirmed. The vaso-dilator effects, dependent on axone reflexes, as described in the paper, were, however, well recognized phenomena.

With regard to the pathways for parasympathetic impulses descending in the cord from higher centres down to the sacral segments, no definite tracts were described. The voluntary control of the bladder and the rectum was probably effected indirectly as described in the paper, namely, by the contraction of the abdominal wall, diaphragm *et cetera*. Once started, the organs tended to evacuate completely.

The only parasympathetic nerves of which Professor Wilkinson knew as existing in relation to the thoracic and lumbar viscera were those which descended via the vagus. The cranial outflow of the parasympathetic was believed to exert a control on the alimentary canal and derivatives, as far down as the centre of the transverse colon, and the pelvic outflow exerted an influence on the rest, down to the anal canal.

In reply to Dr. Burnell's question, Professor Wilkinson stated that Brown relied entirely on the protein shock method of raising the body temperature, but that he had described the method used by Gask and Ross because it was based on the latest researches of Lewis, and he thought that the audience would be less familiar with it and would be interested to hear of it.

Correspondence.

THE MORTALITY OF APPENDICITIS.

SIR: I have read with very great interest the paper contributed to your journal (May 25) by Dr. Colvin Storey, and must congratulate him on his splendid low mortality rate. To have been able to operate on 898 cases of appendicitis with only eleven deaths is something indeed to be proud of.

There are, however, a few points that I think Dr. Storey might have dwelt upon that could have been made very instructive; he might have taken the Royal Prince Alfred Hospital's fatal cases and have made an effort to show the relationship of the rate of mortality to the day of the attack on which the case was operated.

It is a well known fact that the rate rises very suddenly if acute appendicitis cases are operated on during the third, fourth or fifth day from the onset of the first distinctive symptoms that indicate the acute trouble. It would be, therefore, of very great interest to find out the days since the onset of the attack that the cases were operated on that gave the mortality rate of from 38% to 48%. There are three points in connexion with acute appendicitis that have a fascination for the expert surgeon: (a) the day of the disease on which the operation was performed; (b) the taking away or the leaving behind of the diseased appendix in peri-appendicular abscess cases; (c) the comparison, of the mortality rate, between those acute cases in which immediate operation is performed with the results in those cases where the Ochsner or Sherren expectant treatment has been followed.

These are really the interesting and important points in connexion with acute appendicitis, when that disease is in the hands of expert surgeons. For we know that the subacute catarrhal cases and the old chronic cases should have no operative mortality, except when such complications as ileus, pseudo-ileus, pneumonia or pulmonary embolism are unkind enough to take a hand in the after-treatment. The mortality of acute appendicitis is chiefly the mortality of diffuse septic peritonitis, and, as we have not advanced in the actual treatment of that condition in the last fifty years, we cannot pat ourselves on the back; all that we have done, thanks to Lister, is to prevent. We almost always fail once we get into holts with the implacable foe of the abdominal surgeon. This no doubt is the explanation of the terrible mortality rate given in Dr. Storey's statistics of the Royal Prince Alfred Hospital. It is to be hoped that the new oil emulsion treatment will be the real saviour of the abdominal surgeon.

I think there is one passage to which objection may be taken without being accused of captiousness. Dr. Storey says:

I need not remind you that the initial pain is due to tension in the appendix and is referred to the branches of corresponding intercostal nerves. It is usually felt near the umbilicus, but may be experienced anywhere in their distribution.

Now I beg to say that this statement is not correct. The first pains that arise in a case of acute appendicitis are diffuse visceral pains, often experienced in the epigastric or umbilical region, but they are not referred pains and have nothing whatever to do with the branches of the intercostal nerves. These first visceral pains are due to the inflammation in the appendix causing increased tension (?) and the impulse generated in the appendix is then conveyed by the sympathetic splanchnic nerves to the superior mesenteric plexus, where the impulse is distributed to the intestines, and it is felt as a diffuse visceral pain somewhere in the epigastric or umbilical region. It is not until the parietal peritoneum becomes involved some hours later that the eleventh and twelfth thoracic nerves are affected. Mackenzie showed that the pain from some of the viscera was conveyed to the spinal cord and might there create an "irritable spinal focus", as he termed it; but in recent years the more general

opinion is that the afferents from the abdominal viscera, including the appendix, are apparently unable to set up an irritable spinal focus, and so it is not until the parietal peritoneum is sufficiently irritated or inflamed that we get the pain "referred to the intercostal nerves", as stated by Dr. Storey.

The explanation of this referred pain is that the parietal peritoneum is supplied by somatic nerves and, as there is a reflex arc commencing either in the peritoneum itself or in the loose connective tissue lying just outside the peritoneum, as Ramstrom was the first to point out, and as these nerves communicate with the cord, the reflex causes the impulse that is sent down the eleventh and twelfth thoracic nerves, and sometimes the first lumbar nerve, to be registered in the right lower quadrant, and we get local pain, hyperæsthesia of the skin, hyperalgesia of the muscles and muscular rigidity. But this may not take place if the appendix happens to be in a silent area or what Zachary Cope calls "non-demonstrative areas". Such an area would be where we have a retrocaecal appendicitis, or where the appendix is well down in the true pelvis; for in the latter case the peritoneum lining the true pelvis is situated too low to be segmentally represented by the abdominal muscles. And so we find, as is often the case with children who are prone to have retrocaecal appendicitis, that an examination of the right lower quadrant fails to show the usual physical signs of hyperæsthetic skin and hyperalgesic muscles. Later on, however, when the parietal peritoneum becomes generally involved, we may get muscular hyperalgesia and rigidity in the region of McBurney's point and elsewhere in the right lower quadrant.

As Dr. Storey has remarked, we may get help in diagnosing these cases by a rectal examination, especially in children; and also by vaginal examination in women.

Dr. Storey may feel consoled by the following bloomer that appears in the last (eleventh) edition of Howell's "Physiology", when he says: "It is interesting that affections of the serous cavities—e.g. the peritoneum—do not cause reflected pains or cutaneous tenderness as in the case of the viscera." Samson Wright (1934) knows better, for he says: "But when as a result of disease the parietal peritoneum is involved, somatic afferents are stimulated which give rise to referred superficial pain, tenderness, and (reflex) rigidity."

Yours, etc.,

W. J. STEWART MCKAY.

Lismore,
New South Wales,
May 26, 1935.

PROPRIETARY REMEDIES FOR INJECTION.

SIR: During recent years physicians have been inundated with literature and samples of so-called non-specific remedies for treatment of chronic joint and chest conditions, for acute infections, such as pneumonia, and for pyrexias of unknown origin. It seems high time that the position regarding these should be carefully examined and that the correct attitude of the profession towards them should be clearly defined.

There is no doubt that non-specific therapy proves an amazingly useful weapon at times, although failures are far more common than successes. Probably vaccines of various sorts, peptone and sterile milk are the best recognized agents used. We have gradually come to recognize in which fields they are most useful and how they are likely to affect the patient.

These are agents of known composition, and their sterility is assured by standard methods of preparation. Very different, however, is the position of certain proprietary remedies. These are sent to us in elaborate packages with literature making quite extravagant claims for their efficiency and complete harmlessness. Almost invariably the composition is kept secret and vague statements are made about "denatured proteins" or "antigenic substances" which mean next to nothing.

The point which I wish to make is that non-specific therapy will only become really useful after years of careful observation. If agents of known composition are used, we will gradually learn how to use them effectively, but even then it is bound to be a slow and toilsome business. If the agents used are of mysterious origin, any real gain in knowledge will be quite impossible, although we will certainly enrich some of the firms producing them.

Apart from this, there is the responsibility to the patient. Generally he trusts implicitly to our good sense and integrity, and there is no doubt that there would be a serious scandal if it became known that physicians were silly enough to inject concoctions of unknown origin simply on the assurance of some rather obscure firm.

It is interesting to imagine the legal proceedings if illness or death resulted after one of these remedies. The first questions to be asked would be regarding composition, sterility and possible effects of the agent. The physician's plea that it was a businesslike bottle with a nice looking label and laudatory literature would hardly compensate for the fact that he knew nothing of the contents.

My experience is that some of these reagents are not by any means harmless and, even if ill-effects occur only in those with some idiosyncrasy, it should give us some pause.

I would suggest that some authority should be established which could examine each of these remedies, and that the profession should refrain from using them until this authority was satisfied as to the composition, sterility and harmlessness of the agent. Some estimate might also be made of its usefulness. Such an examination would require skilled work, and this should be adequately paid for by the firm concerned.

Admittedly there are objections to these suggestions. We cannot wait for a complete elucidation of a drug's action before using it, and no doubt it would occasionally delay the utilization of some really useful remedy. But it would enable us steadily to build up this part of our knowledge and prevent futile expeditions which will lead us only further into the fog.

Voltaire remarked that "a physician is a person who pours medicine, of which he knows little, into a body of which he knows less". Surely in this age of hypodermic therapy we must take care lest we deserve this jibe more than ever.

Yours, etc.,

"TIMOCLES."

May 31, 1935.

MEDICAL HISTORY OF THE WAR.

SIR: For the purpose of the official Australian "Medical History of the War, 1914-1918", I am desirous of obtaining a bibliography of work published by Australians relating to any important aspect of that event, whether scientific, technical or descriptive. A good many reprints and special army reports are held by the Australian War Memorial, originally the Australian War Records Section, in addition to the special reports and memoranda furnished during or soon after the war at the request of the Medical Collator, and appendices to the Official War Diaries. These have been supplemented by search through available files of the medical journals. But the machinery at my disposal is not adequate to insure even reasonable completeness, and it would greatly assist if members of the profession would make known to the Australian War Memorial any of their writings which they believe, or hope, however modestly, may have been of some influence at the time and/or of some significance or service as history.

I venture to ask if you would give publicity to this need through your correspondence columns. Any communication on the matter should be addressed: Colonel A. Graham Butler, A.A.M.C., Defence Department, Melbourne.

I should like, in addition, to suggest that members of the A.A.M.C., A.I.F., should provide for the deposition in the Australian War Memorial of their personal war records—private diary, photographs, letters—all, in fact, of the *omnium gatherum* that at present seems to have senti-

mental or personal value only. Any such could be deposited at once "on loan" and would be available, if required, to the donor at any time.

Yours, etc.,

A. GRAHAM BUTLER.

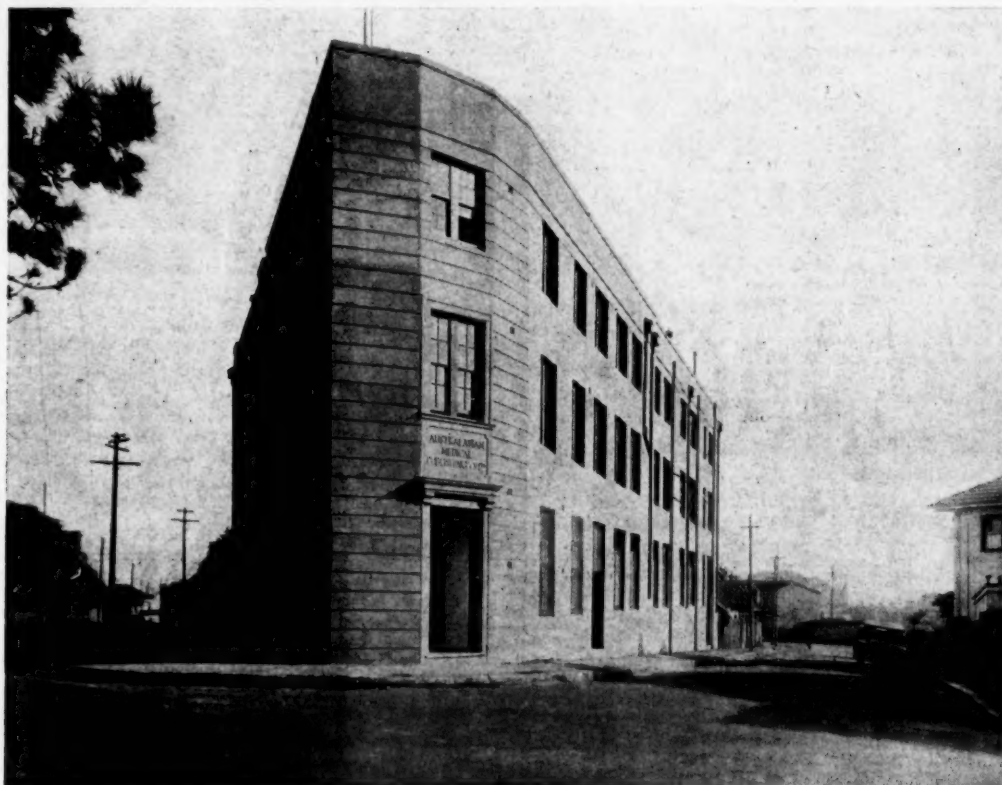
June 12, 1935.

Australasian Medical Publishing Company, Limited.

ADDITIONS have been made to The Printing House, Seamer Street, Glebe, the home of the Australasian Medical Publishing Company, Limited.

and the company will no longer have to employ an outside firm to bind the books that it prints. The managerial offices have been transferred to the new portion of the building on the first floor, and the editorial offices are on the second floor. These offices are finished in polished maple; they are well provided with windows and are of modern design. The composing room has been partly rearranged; the reading room has been transferred to the second floor, in the space previously occupied by the managerial offices; and a new enclosure has been made for the monotype caster. An additional monotype caster has been installed. The floors are all of reinforced concrete and the walls are capable of carrying additional stories.

The manager of the Australasian Medical Publishing Company, Limited, will be glad to welcome any readers of this journal who may wish to visit The Printing House.



The original building, comprising three floors, did not extend over the whole of the land owned by the company. A triangular piece of ground on the corner of Seamer Street and Arundel Street was not occupied. During recent years the need for more room has been felt, particularly in the machine room on the ground floor. At their meeting in August, 1934, the Directors of the Australasian Medical Publishing Company, Limited, decided that if suitable arrangements could be made, and if the time seemed opportune, the building should be completed. In the beginning of this year a start was made with the extension scheme. Plans were drawn by Mr. G. N. Kenworthy, A.R.A.I.A., and a contract was let to Mr. A. C. Eden. The building, as shown in the accompanying picture, has been completed. The extension was designed to conform to the original structure, and a dignified entrance has been provided on the corner of the building which overlooks Parramatta Road. On the ground floor 638 square feet of additional space have been added to the machine room. In this room a bindery has been installed;

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on June 10, 1935.

The following degrees were conferred in person by the Chancellor:

Bachelor of Medicine (M.B.): Bruce Goodwin Hill.

Bachelor of Medicine (M.B.) and Bachelor of Surgery (B.S.): Joseph Christie, Maida Elsie Wilhelmina Hall, Frank Hatcher, William Hessel Kellett, John Victor Latham, John Russ Lee, Stanley Clive Maynard, Allen Percival Prior, and Henry Walter Ritchie Sharp.

The following degree was conferred *in absentia*:

Bachelor of Surgery (B.S.): Egmont Friedrich Hellmuth Theile, M.B.

Books Received.

- THE MATERNITY AND CHILD WELFARE MOVEMENT, by G. F. McCleary, M.D., D.P.H.; 1935. London: P. S. King and Son, Limited. Crown 8vo., pp. 237. Price: 7s. 6d. net.
- RECHERCHES ANTHROPOMETRIQUES SUR LA CROISSANCE DES DIVERSES PARTIES DU CORPS, by P. Godin; Second Edition; 1935. Paris: Amédée Legrand. Royal 8vo., pp. 279.
- THE ESSENTIALS OF MATERIA MEDICA, PHARMACOLOGY AND THERAPEUTICS, by R. H. Micka, M.D., F.R.C.P.I.; 1935. London: J. and A. Churchill. Demy 8vo., pp. 405. Price: 12s. 6d. net.
- CHRONIC NASAL SINUSITIS AND ITS RELATION TO MENTAL DISORDER, by F. A. Pickworth, B.Sc., M.B., B.S., A.L.C.; 1935. London: H. K. Lewis and Company, Limited. Super royal 8vo., pp. 168, with 83 illustrations. Price: 16s. net.
- THE CLINICAL ASPECTS OF VISCERAL NEUROLOGY, WITH SPECIAL REFERENCE TO THE SURGERY OF THE SYMPATHETIC NERVOUS SYSTEM, by W. K. Livingston, M.D.; 1935. London: Baillière, Tindall and Cox. Super royal 8vo., pp. 265, with illustrations. Price: 22s. 6d. net.

Diary for the Month.

- JULY 1.—New South Wales Branch, B.M.A.: Organization and Science Committee.
- JULY 2.—Tasmanian Branch, B.M.A.: Council.
- JULY 2.—New South Wales Branch, B.M.A.: Council (quarterly).
- JULY 3.—Western Australian Branch, B.M.A.: Council.
- JULY 3.—Victorian Branch, B.M.A.: Branch.
- JULY 4.—South Australian Branch, B.M.A.: Council.
- JULY 5.—Queensland Branch, B.M.A.: Branch.
- JULY 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
- JULY 9.—Tasmanian Branch, B.M.A.: Branch.
- JULY 12.—Queensland Branch, B.M.A.: Council.
- JULY 16.—Tasmanian Branch, B.M.A.: Council.
- JULY 16.—New South Wales Branch, B.M.A.: Ethics Committee.
- JULY 17.—Western Australian Branch, B.M.A.: Branch.
- JULY 17.—Victorian Branch, B.M.A.: Clinical meeting.
- JULY 18.—New South Wales Branch, B.M.A.: Clinical meeting.
- JULY 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
- JULY 24.—Victorian Branch, B.M.A.: Council.
- JULY 25.—South Australian Branch, B.M.A.: Branch.
- JULY 25.—New South Wales Branch, B.M.A.: Branch.
- JULY 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser", pages xiv, xv, xvii.

- AUSTIN HOSPITAL FOR CANCER AND CHRONIC DISEASES, HEIDELBERG, VICTORIA: Resident Medical Officer and Pathologist, Resident Medical Officer.
- CHILDREN'S HOSPITAL, CARLTON, VICTORIA: Assistant Resident Medical Officer.
- LAUNCESTON PUBLIC HOSPITAL, LAUNCESTON, TASMANIA: Resident Medical Officer.
- MENTAL HOSPITAL, NEW NORFOLK, TASMANIA: Junior Medical Officer.
- MOUNT ISA MINES, LIMITED, MOUNT ISA, QUEENSLAND: Chief Medical Officer.
- PRINCE HENRY HOSPITAL, SYDNEY, NEW SOUTH WALES: Junior Resident Medical Officers.
- ST. GEORGE DISTRICT HOSPITAL, KOGARAH, NEW SOUTH WALES: Senior Resident Medical Officer.
- THE EASTERN SUBURBS HOSPITAL, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.
- THE RACHEL FORSTER HOSPITAL FOR WOMEN AND CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Medical Officer (female).
- THE UNIVERSITY OF SYDNEY, NEW SOUTH WALES: Liston Wilson Fellowship.
- THE WOMEN'S HOSPITAL, CROWN STREET, SYDNEY, NEW SOUTH WALES: Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Chillagoe Hospital. Members accepting LODGE appointment and those desiring to accept appointments to any COUNTRY HOSPITAL, are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	Officer of Health, District Council of Elliston. All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor", THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £3 for Australia and £2 5s. abroad per annum payable in advance.

ce.

any
ving
nch
the
C.I.

ddly
Die-

ted
en-
ey.
Dis-
any

ro-

lde

es'

nt-
ept
tY
wn
eir
ere

of
s-
in

in

n,

t
n
e

,
b,
:

,
s,
v,
k,
t,
y,
y,
i